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N.B.—The Society does not hold itself in any way responsible for the statements made or the views put forward in the various papers.

Section of Obstetrics and Gynæcology

President—GILBERT I. STRACHAN, M.D., F.R.C.P., F.R.C.S., F.R.C.O.G.

[*January* 25, 1952]

THE following specimens were shown:

Carcinoma of the Body of the Uterus.—Dr. N. A. RICHARDS.

Secondary Carcinoma in Rectovaginal Septum.—Dr. G. B. LIVINGSTON.

Secondary Carcinoma of the Vagina; Primary in the Sigmoid Colon.—Dr. H. P. FERREIRA.

Carcinoma of the Clitoris with Blood-borne Metastases.—Dr. R. M. FEROZE.

Inoperable Adenocarcinoma of the Ovary, Becoming Operable after Deep X-ray Therapy.—Dr. O. C. SANDYS.

Pyometra in Half of a Double Uterus.—Mr. C. H. DE BOER.

Hydatid Cysts of the Pelvis.—Dr. V. A. CROXFORD.

An Ectopic Pregnancy in a Tuberculous Fallopian Tube.—Mr. E. COPE.

Gross Post-Menopausal Œstriosis with a Small Thecoma.—Dr. A. AMBERY SMITH.

Struma Ovarii.—Mr. ARTHUR WILLIAMS.

Endometrial Sarcoma.—Dr. D. PELLS COCKS.

Stromal Endometriosis.—Dr. A. E. FYFE.

[*February* 22, 1952]

The Association of Radium and Surgery in the Treatment of Carcinoma of the Cervix

By DAVID W. CURRIE, F.R.C.S., F.R.C.O.G.

IT is interesting to note the changes which have taken place in the treatment of carcinoma of the cervix during the last thirty years. When I was a student in 1926 Mr. Carlton Oldfield was attacking the disease by surgery using both an abdominal and an abdomino-vaginal approach. In 1932 he and his successor in the Chair were treating all their cases with radium. A similar change was taking place all over the country and in some centres the gynæcologist had even lost the treatment of the disease altogether. Because of the surgical interest afforded by pelvic dissection, I still made occasional excursions into the forbidden territory during the years 1936 to 1939. At the beginning of the war radium was not available to us, so that from 1941 onwards, being loath to give up a radical hysterectomy, I combined radium with surgery in the treatment of carcinoma of the cervix. The original reason for this association of radium and surgery in the treatment of carcinoma of the cervix was, therefore, in my case, most haphazard.

Other surgeons I know have been working along these lines. Schlink and Chapman (1944) recorded the results of 112 cases in 1944 and later, in 1950, Schlink again described his results, this time quoting 246 patients treated by radium and surgery, with 54% surviving five years and 51·3% ten years. I take this opportunity of relating my experiences in the hope that they will excite criticism.

In June 1936 I operated upon my first case of carcinoma of the cervix and since that date I have operated upon 122 patients, all of whom have been discharged from hospital alive, except one. This was a late growth treated in 1940 when we had no radium. She died of a low-grade anaerobic infection ten weeks after the operation. This report will deal with 103 cases, a consecutive group, the 19 others were treated after the tables were compiled.

Choice of Patient

Suitability for operation was determined by assessing the state of the cervical growth and then the patient's general condition. Since radium alone affords a fair chance of cure it seemed wrong to risk the operation in those patients suffering from any concurrent cardiac, respiratory or renal disease. Age and adiposity were not considered to be deterrent factors. The bulk of my cases fall into the first two groups, but as the years have passed I have operated on more advanced disease. Examination under an anæsthetic is possible when the radium is being inserted and this is the time when, after consultation with the anæsthetist, we decide which course of treatment we shall follow. Such a decision is by no means final, and in quite a number of cases we have changed our minds when inserting the second dose of radium, and subsequently removed the uterus.

The ages of the patients operated upon were as follows:

21–30 years	2
31–40 years	26
41–50 years	36
51–60 years	31
61–70 years	8

Since 1943 I have operated upon 55% of all the patients I have seen. This is a high figure for the present day; it is so because many early growths have been referred to me by the Radiotherapy Department as being suitable for surgical removal. This is an opportune time to pay tribute to those members of the Leeds Radiotherapy Centre with whom I have been working in the closest liaison.

The radiotherapeutic department uses the Stockholm technique: three separate insertions of radium, each insertion consisting of 40 mg. of radium into the uterus and 40 mg. into each lateral fornix, left in situ for twenty-four hours. The majority of my patients were given one dose equivalent to 2,880 mg. hours. Some have had two doses and a few the full radium treatment. The operation was undertaken three weeks after the insertion of radium. I believe that the pre-operative radium cleanses the growth and greatly reduces the danger of infection. I think it reduces the amount of bleeding encountered during the operation and may help to prevent recurrences in the vaginal vault. I have not seen any severe sepsis following the operation; by that I mean the complete absence of localized pus formation and blood infection. The one case referred to already was not given any radium, and another which developed a mild paralytic ileus responded rapidly to treatment. Naturally, other factors such as the increasing use of antibiotics play an important if not dominating part. I have not, as yet, seen a recurrence in the vagina and I wonder whether the radium may be of help here. In many cases the radium kills the cancer of the cervix so that by the time the operation is undertaken the surface, as well as being clean, is no longer friable and so there is less danger of dissemination by trauma. The radium may also destroy the growth spreading down the fascial planes of the vagina but as it is my custom to remove at least 5 cm. of the vagina, this may be an insignificant point.

I have frequently toyed with the idea of giving more than one dose of radium. I feel that the actual value of the pre-operative radium is limited and is not enhanced by giving a major dose. Schlink eventually gave a dose of 7,000 mg. hours, a dose which is now considered adequate for full radium treatment. What is the value of subjecting the cervix and surrounding organs to this dose of radium if a short time afterwards we intend to remove the uterus altogether?

In his table of post-operative morbidity Schlink describes quite a number of complications which could be attributed directly to the effect of the radium such as injuries to the bladder, rectum and ureters. I submit that if the action of radium is limited and has no effect upon the lymph nodes it is wrong to use any dose large enough to cause complications, however infrequent these may be. I do not think that repeated doses make the operation more difficult; more important is the lapse of time from the first insertion to the operation. The longer the interval the more difficult did I find the dissection to be, especially at that crucial point where the uterine artery crosses the ureter. By a process of experiment, three weeks was chosen and adhered to. Brunner (1932) said that a marked leucopenia

occurred at this time and that the bacterial virulence was at its highest but the outstanding clinical fact remains that sepsis was noticeable by its absence. I have, however, noticed the presence of large non-malignant lymph nodes at the operation, but rarely the surrounding œdema so often described.

The effect of radium on the primary growth was recorded by Dr. Norman Lissimore in 52 cases. Four grades were distinguished. In the first no sign of living growth could be discovered when the uterus was subsequently sent for examination; in the second, gross necrosis with few living cells remaining; in the third, some necrosis but still many foci of living growth while in the last the radium had either no effect whatever or so little as to be scarcely noticeable. In spite of the fact that in most cases only one-third of the usual dose of radium was given, many primary growths had disappeared altogether within three weeks and the cervix was clear. The percentage of these grades was as follows:

Destroyed	17·3%
Good effect	32·7%
Poor effect	40·0%
Indifferent or no change ..	10·0%

In one case where the operation was conducted five months after a complete Stockholm treatment, many islets of living cancer were found embedded in a fibrous cervix. During the last twelve months 3 patients considered at first to have too extensive growths for surgical removal were returned to me by the Radiotherapeutic Department after complete radium treatment. They all had unaffected malignant glands, but no growth was found in the cervix; in not one single instance did the pathologist find any effect of the radium on malignant nodes. Schlink and his colleagues Chapman and Chenhall were impressed by similar findings. In a much larger series of pathological investigations they came to the conclusion that radiotherapy does not entirely eliminate all cancer cells from the cervix, however well-healed the surface may be, and it has no effect whatever on lymph glands invaded by squamous or adenomatous cancer cells.

Gland Involvement

Lymph glands were involved in 26 patients. This is a high figure bearing in mind that only early growths had been shown. Read (1948), in his Address to the New York Obstetrical Society in 1948, assessed the involvement of glands as follows:

Stage 1	20 to 25%
Stage 2	30 to 35%
Stage 3	40 to 50%
Stage 4	Over 60%

In Victor Bonney's series (1941), 40% had glandular involvement and, as he pointed out many years ago, even the smallest primary growth on a freely mobile cervix may have spread to the glands and there is no clinical method yet known which can demonstrate this spread. The glands most often affected were those lying in the angle between the external and internal iliac arteries. The obturator glands were more rarely involved and the so-called Wertheim gland on the pelvic floor was only isolated as a malignant node on one occasion.

25% of this series had malignant glands, none of which was affected, and if the best results imaginable could be obtained by the use of radium as applied at present, i.e. the destruction of every single primary growth, then the optimum cure figure could not be greater than 75%. But the power of radium is much less than this and some growths are not even touched by it. In a series such as this we should estimate that radium alone offered a chance of curing, at most, only 60% of the patients. Surgery must be considered still as an important means of curing cancer of the cervix provided that the surgical procedure in itself is not associated with too high a mortality. With the use of pre-operative radium, penicillin, sulphonamides, blood transfusion, the marked improvement in anæsthesia and, above all, a careful surgical technique, the mortality of a wide and radical excision can be reduced to that of a simple panhysterectomy. Read and Cook lost only 6 patients in 207 cases and Schlink 18 in 417.

The Operation

Prior to the operation a blood transfusion is given if the hæmoglobin is below 55%. The patients are now admitted four days before operation for a prophylactic dose of penicillin and vaginal cleansing. Dr. R. Lawrence, my anæsthetist, sees all the patients before operation and is responsible for the intravenous therapy as well as the anæsthetic. He usually prefers to start the operation with an intravenous

glucose drip changing over to blood as he thinks fit. He uses pentothal induction followed by gas, ether and oxygen with a relaxing drug and, of late, has been experimenting with the hypotensive technique.

The vagina is cleansed, packed with gauze soaked in flavine and a self-retaining catheter inserted into the bladder. The abdomen is opened through a left paramedian incision and the intestines packed off. The uterus is controlled by two Bonney's clamps placed vertically on the broad ligament on either side of the body. I remove the glands early, clearing them off the blood vessels and sweeping them internally towards the uterus, having first freed the ureters and the bladder. I thought that I had devised this method but recently I saw a film, produced twenty years ago by Mr. Aubrey Goodwin, showing the identical procedure which he has been carrying out for many years. The round ligaments are divided near the pelvic brim, the peritoneum of the utero-vesical pouch is incised and the bladder pushed well down. The peritoneum on both sides is then incised along the pelvic brim from the round ligament to the bifurcation of the common iliac artery and the ovarian pedicle clamped and divided here. The tubes and ovaries are then cut away and removed altogether. The ureters are then isolated and freed from the pelvic brim as far as the ureteric canal. This action strips up a flap of peritoneum from off the pelvic wall and a broad rectangular exposure of the side walls of the pelvis is obtained if this flap is divided vertically down to the utero-sacral ligament on the pelvic floor.

The fascial sheaths of the external and internal iliac vessels are then opened. The former along its whole length and the tissue in the angle between the vessels freed and pushed forwards until the obturator nerve is exposed. All the fatty tissue containing the glands about the arteries and in the region of the obturator foramen can now be stripped out by carrying the finger forwards and bringing it up between the bladder and the obliterated hypogastric artery. The artery is now divided between clamps at the pelvic brim and the proximal part traced back to the origin of the uterine artery. This artery is likewise divided between clamps and by the simple manœuvre of lifting it inwards the ureter is exposed and freed in the ureteric canal. The ureter is then freed with ease as far as the bladder. This simple manœuvre completed in little more than five minutes, leaves the pelvic wall and adjacent pelvic floor completely clean, preserves the vesical vessels, isolates the ureter with safety and is accompanied by a minimal amount of bleeding. The posterior attachments of the uterus have not yet been disturbed, they are now divided between clamps as also are the cardinal ligaments. The remainder of the operation follows along the usual lines. I close the vagina with a purse-string suture, having first thrust a piece of gauze down from above and leaving a wick sticking out to drain the cellular space. After spraying this space with penicillin and sulphathiazole powder the pelvic peritoneum is closed. The average time for the operation is sixty to sixty-five minutes. Penicillin and sulphamerazine are given for three days. The pack and catheter are removed in forty-eight hours.

Results of the Operation

Of the 103 patients recorded there was no immediate post-operative death. 11 have since died, 1 ten weeks after the operation, a further 8 within twelve months and 2 nearly five years later. The remainder, apart from 2 who have emigrated and are still alive, are under constant supervision. A few not followed up by me personally are seen at the Radiotherapy Centre. A notable feature is the regularity with which these patients attend and especially the happier mental outlook of the patients treated by surgery than by radium alone. During the first two years they attend every three months and then every six months for three years and afterwards once per year.

We were unable to discover the cause of death in all the cases but when possible a post-mortem was undertaken. In 1 case only was a pelvic growth discovered. 1 had abdominal carcinomatosis, 2 isolated abdominal masses, 1 secondaries in the lung and brain, 1 brain and spine and another brain only. All these patients died within twelve months of the operation and I think we are justified in thinking that the disease had spread far beyond the field of operation at the time this was undertaken.

I have been particularly fortunate in not losing a patient from pulmonary embolus. I do not use a high Trendelenburg position nor do I drop the foot of the table much, but it is interesting to note that whereas I allow all my other patients to get up at an early date I keep my Wertheim hysterectomies in bed twelve to fourteen days. There is no special reason why this course should be followed. Probably the ward Sister prefers to keep them in bed.

One patient, a nullipara, died from a perforated carcinoma of the splenic flexure. She was operated upon but without success; the pelvis was clear of growth. 2 other women have developed carcinoma of the colon after the removal of the uterus and were both treated successfully. This association of cancer of the cervix with other independent primary growths was referred to by Victor Bonney, and in my series, apart from those mentioned, there was a primary adenocarcinoma of the ovary and a myosarcoma of the uterus. These patients are alive and well.

Complications of the Operation

Post-operative complications have been rare. Major sepsis was met with only once although late pelvic cellular infection was noticed twice, as long as three months after the operation. These responded well to short-wave diathermy. In one of these the symptoms were so acute that a provisional diagnosis of acute appendicitis had been made. 2 patients only had catastrophes related to the urinary tract. In 1, a left ureteric fistula developed three days after the operation which necessitated a nephrectomy four weeks later. The kidney showed marked hydro-nephrosis in that short time. The other developed low abdominal pain six weeks after her discharge and a local surgeon incised the vault. I saw her some three weeks later and successfully repaired a large vesico-vaginal fistula. I can only think that she had had a necrosis of the bladder with extravasation of urine. It is my custom to free but not cleanse the ureter along the greater part of its pelvic course and although this procedure is contrary to the advice of other surgeons and not in keeping with the recent publication of Dudley Racker (1951) on the blood supply to the ureter, I can only record one case of known damage to this structure.

Results of Treatment

I have calculated the results up to January 1, 1952, and they show that the percentage of relevant cures after five years is 79·5% and after ten years 66%. 10 out of 15 operated upon more than ten years and 35 out of 44 more than five years are still alive.

		Still alive	% survival rate
No. of patients operated upon five years	44	35	79·5
No. of patients operated upon ten years	15	10	66·0

The figures of the Leeds Radiotherapy Centre for stage 1 and stage 2 cancers treated between 1931 and 1940, and calculated according to the League of Nations criteria, were 38·4% and 30·5%.

Conclusions

The association of radium with surgery in the treatment of carcinoma of the cervix has a very limited value. If used at all it must be used prior to the operation. To me there seems very little doubt that it cleanses the cervix and diminishes the incidence of infection, it may also prevent local recurrence. The most important value of the association has been in the material it has provided for the pathologist. We notice how varied is the reaction of each individual growth to the radium and how the glands frequently invaded even in the earliest growths are not touched by the radium with the technique and screenage used at the present day. This leads us to the conclusion that if the results of our treatment are to be improved we must have recourse to surgery, and if an operation is possible the more advanced the growth the more need is there for surgery since the more likelihood is there of glandular involvement. But surgery must not be entertained if the mortality of the operation is high or else its value is rapidly nullified. This calls for an especial education of the surgeon in the technique of the operation and expert skill from all his assistants.

(A film was shown to demonstrate the operative technique on carcinoma of the cervix.)

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The following speakers took part in the subsequent discussion: **Mr. Leslie Williams, Mr. Charles Read, Mr. J. B. Blaikley, Dr. C. G. Roworth, Mr. J. V. O'Sullivan, Mr. A. H. Charles** and **Professor G. I. Strachan.**

Stump Carcinoma

Its Treatment and the Relation between Parity and Incidence

By T. F. Redman, M.B., F.R.C.S.Ed., M.R.C.O.G.

It has been amply shown in the literature on cervical cancer that the incidence of carcinoma in the stump is as high if not higher than that in the cervix of the intact uterus and that the case for panhysterectomy is well established.

I shall consider two aspects of stump carcinoma: the incidence in nulliparous women and the treatment of the condition.

Parity

In connexion with nulliparity Table I gives the findings of three authors. The series show remarkable agreement and, taken as a whole, give the figure of 22% for the incidence of nulliparity among cases of stump carcinoma. But this figure by itself means nothing, nor does the corresponding figure in ordinary cervical carcinoma: it is necessary to consider the incidence of nulliparity in the "population" from which the condition is drawn. Table II shows the incidence of nulliparity in various groups of women.

TABLE I.—STUMP CARCINOMA IN NULLIPAROUS WOMEN

Author	No. in series	No. of nulliparous patients	Per cent
von Graff (1934) Collected	300	67	22
Meigs (1936)	26	6	23
Fricke (1939)	108	23	21
	434	96	22

TABLE II.—THE INCIDENCE OF NULLIPARITY

Intact uterus	(1) All women over 34 years old	16·7% (70,000)
	(2) Cases of ordinary cervical carcinoma..	4·7% (1,535)
After S.V.H.	(3) Women with a cervical stump	37% (100)
	(4) Cases of stump carcinoma	22% (434)

Standard error of the difference between (3) and (4) = 5·2

(Figures in brackets show the size of the series)

(1) The figure for the incidence of nulliparity in women over 34 is obtained from the Registrar-General's Statistical Review of England and Wales (1949).
(2) The incidence of nulliparity in ordinary carcinoma of the cervix is quoted from three large series by Spencer (1932), Healy (1934) and Maliphant (1949). (3) The percentage of women with a cervical stump who are childless was found as a result of examining the notes of 100 consecutive operations of subtotal hysterectomy at St. Mary's Hospitals, Manchester. (4) From Table I, the higher incidence of nulliparity among the "stump population" is to be expected in view of the fact that supravaginal hysterectomy is performed most often for conditions which are associated with sterility. In the case of ordinary cervical carcinoma, the predisposing influence of child-bearing is apparent without any statistical test, but in the case of stump carcinoma, the effect of parity is less obvious: the difference is significant but not dramatically so. The practical application of this is to weaken the argument: "this woman has a nice healthy nulliparous cervix, it is safe to perform supravaginal hysterectomy." Nulliparity does not offer the same degree of protection against carcinoma in the woman with a cervical stump as it does in the woman with an intact uterus—another argument against supravaginal hysterectomy.

Treatment

The use of radium (Table III)—by far the most favoured method—has produced a 5-year salvage of 25% for "true" stump carcinoma in the composite series. Incidentally it will be noted that the results with the "coincident" variety, viz. 16%, are not so poor as has been frequently stated in the literature, e.g. by Nuttall and Todd (1936), in fact, the results with "true" stump carcinoma are not

TABLE III.—CARCINOMA OF THE UTERINE STUMP TREATED BY RADIUM

Author	"Coincident" Cases	"Coincident" 5-year survival	"Coincident" Per cent	"True" Cases	"True" 5-year survival	"True" Per cent
Arneson (1934)	—	—	—	64	9	14
Nuttall and Todd (1935)	18	0	0	8	4	50
Fricke (1939)	42	9	21	57	15	26
Hurdon (1942)	5	3	—	13	10	77
Gemmell (1942)	—	—	—	5	1	—
Donnelly and Bauld (1949)	—	—	—	20	3	15
	65	12	18	167	42	25

Standard error of the difference = 5·8

(A "coincident" carcinoma is one which occurs within 2 years of the original hysterectomy and is presumed to have been present at the time of operation.)

TABLE IV.—CARCINOMA OF THE UTERINE STUMP TREATED BY SURGERY

Author	No. treated	Alive in 5 years	Method of treatment
Wertheim	8	4	
Peham and Amreich (1930)	13	6	Vaginal removal
Meigs (1936)	1	0	Wertheim
	2	1	Vaginal removal
	24	11	

significantly better. Surgery (Table IV) has been less favoured except in the Viennese School and few figures are available, yet the composite series produces a five-year salvage of about 50%, which compares not unfavourably with the radium results, although the surgically treated cases are too few and the information too scanty for firm deductions to be drawn.

Table V shows the results in a new series of stump carcinoma treated at the Christie Hospital, Manchester, between 1935 and 1945. I would like to thank Miss M. C. Tod for permission to use these figures and Miss Russell of the Statistical Department of the Hospital for her help in compiling them. The results are not dissimilar from those achieved with ordinary carcinoma of the cervix at that hospital, viz. 36% absolute five-year salvage. Nor does the technique differ materially, except in

TABLE V.—CARCINOMA OF THE UTERINE STUMP TREATED BY RADIUM

(Christie Hospital, Manchester, 1935–1945)

	No. treated	Alive in 5 years	5-year survival rate
"True"	43	16	37%
"Coincident" .. .. (Found within two years of operation)	16	5	31%

Standard error of the difference = 14

the absence of the full intracavity dosage. An attempt is made to insert 2 units (i.e. 13·33 mg.) into the remnants of the canal and the usual vaginal ovoids are used. Miss Tod says that her main difficulty lies in getting adequate dosage due to the contracted vagina hampering the insertion. Again it is shown that the prognosis in the "coincident" type is reasonably good, in fact the results are not significantly different from those of the "true" variety.

A further point of interest in this series is that there was no recorded case of post-radiation fistula. (This can be compared with a 1·8% incidence of such fistula following radiation therapy of ordinary cervical carcinoma at the same hospital.) One might well expect a relatively high incidence of fistula and indeed Souttar (1934) went as far as to say that radium should not be used, while Meigs (1936) produced 5 fistulæ in 18 cases treated. These good results from the Christie Hospital present a formidable challenge to other forms of treatment.

But has surgery any place, either alone or in combination with radium? In the treatment of ordinary cervical carcinoma the trend is towards the use of surgery in selected cases, and we should reconsider its use for stump carcinoma also. It is likely that in this country the abdominal approach would be favoured and Professor G. Gordon Lennon has given me permission to present a case treated recently on the Professorial Unit at Bristol. This nulliparous patient had had a subtotal hysterectomy in 1939, twelve years previously, for a fibroid which on pathological examination showed early sarcomatous degeneration. There had been no further complaints until December 1950 when intermittent vaginal bleeding began, and on September 18, 1951, nine months after the onset of symptoms, the patient attended our clinic when carcinoma of the stump was diagnosed. She was then 59 years old. A biopsy showed the growth to be an adenocarcinoma, and it was assessed clinically as Stage I. Three applications of radium were given, 2 boxes of 25 mg. being used (the vagina would not hold 3). Seven days after the last dose the stump with the residual tubes and ovaries was removed by a modified Wertheim technique. The most difficult part of the operation was freeing the pelvis from adhesions to bowel caused by the old operation. Later examination of the glands showed both R. and L. obturator glands to be heavily infiltrated with growth although the iliac glands were free. Two months after operation, December 1951, she was fit and well. Our operative experience in this case was like that of Nevinny (1930).

The same criteria of fitness for operation would apply as in the case of the ordinary cervical carcinoma. The adenocarcinoma with its relative resistance to radium may be better treated by operation, as in the case just described. The combined use of radium and surgery may, perhaps, prove to be best, not only in stump carcinoma but in cervical carcinoma in the intact uterus. Certainly radio-therapy is usually a failure when the glands are involved. There is no reason why the Wertheim type of operation should not be performed on the stump, and for similar indications to those which apply in ordinary cervical carcinoma. It seems reasonable to review the method of attack on the stump carcinoma at this time, for within a generation it is hoped that few stumps will exist to turn cancerous and the opportunity for assessing the results of treatment will have been lost.

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The following speakers took part in the subsequent discussion: **Mr. Charles Read, Mr. Ivor Hughes, Mr. J. V. O'Sullivan, Mr. W. R. Winterton, Professor G. I. Strachan, Mr. Leslie Williams, Mr. J. B. Blaikley, Mr. C. M. Gwillim** and **Professor W. C. W. Nixon.**

LIST OF BOOKS RECEIVED FOR REVIEW

(As no reviewing is undertaken in the "Proceedings" this list is the only acknowledgment made of books received for review.)

Industrial Welfare Society. King George VI and Industry: a tribute. pp. 12. London: Industrial Welfare Society. 1952.

Ministry of Supply. Harwell—the first five years. This book gives the first official account of British Atomic Energy Research since 1945. pp. 128. London: H.M.S.O. 6s. 0d. 1952.

BOOKS RECENTLY PRESENTED AND PLACED IN THE SOCIETY'S LIBRARY

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Section of Endocrinology

President—A. W. SPENCE, M.D., F.R.C.P.

[*February* 27, 1952]

DISCUSSION ON RADIOIODINE THERAPY IN THYROID DISORDERS [*Abridged*]

Dr. E. E. Pochin: *Radioiodine Therapy in Thyroid Disease.*

Based on work undertaken on behalf of the Medical Research Council by N. B. Myant, B. D. Corbett, A. J. Honour and E. E. Pochin, from the Department of Clinical Research, University College Hospital Medical School, London.

The radioiodine therapy of thyroid disease is, I think, particularly ready for discussion here because, both in hyperthyroidism and for thyroid carcinoma, the technique of treatment is becoming clear, whereas the methods of selecting suitable cases are much less well established. I think it will be valuable if we can clarify the scope and importance of both these forms of therapy, and how they should fit in to the general management of patients with Graves's disease or cancer of the thyroid.

The principles of these forms of radioiodine therapy are simple. Since radioactive iodine is chemically identical with ordinary iodine, it will be selectively concentrated by the tissues which normally concentrate iodine and there will produce an intense and local beta radiation proportional to its degree of concentration. Moreover, the intensity of radiation will decrease very rapidly at distances of a millimetre from any such iodine-concentrating tissue. By giving a suitable dose of radioiodine, therefore, thyroid tissue can be selectively and strongly irradiated with little irradiation of the body or adjacent structures. And similarly, such thyroid carcinomata as concentrate iodine can be irradiated and possibly destroyed in this way, provided that their cells take up iodine sufficiently strongly to receive a destructive dose of radiation without serious radiation effects occurring first in the rest of the body.

Radioactive iodine is given orally as a dilute solution in the form of iodide. In essence, therefore, the radioiodine treatment of Graves's disease consists in giving what to the patient is a drink of cold water, which is simply repeated if her thyrotoxicosis is not abolished. It offers a treatment which combines the attractions of thiouracil in avoiding operation, with those of surgery in getting the job over and avoiding prolonged supervision. We are dealing, therefore, with an apparently safe and simple treatment, and are not even concerned with its effectiveness, since it is only ineffective if insufficient radiodine has been given. We are concerned with any complications of what may otherwise become the treatment of choice for Graves's disease.

Let me deal with the more theoretical disadvantages first. The associated irradiation of the whole body is slight, and produces no symptoms or blood changes, such as occur with the ten times larger doses given for thyroid carcinoma; nor do local irradiation effects in organs other than the thyroid appear to be significant. The plasma receives only about one-tenth of a roentgen per millicurie while the radioiodine is circulating as iodide, and about 5 roentgens per millicurie during its subsequent circulation in thyroxine combination, most tissues receiving considerably less than does the plasma. Significant doses could only be received by organs concentrating thyroxine highly, and the ovary is unlikely to receive amounts of radiation greater than in some routine diagnostic procedures. It should, however, be noted that the fœtal thyroid, or that of a breast-fed child, will share the dose, and the treatment is inapplicable after the 16th week of pregnancy.

The thyroid may occasionally become slightly tender but we have only seen severe tenderness or associated œdema of surrounding tissues at the much higher doses used for total thyroid destruction. There is no good evidence of damage to adjacent structures at either dose level. We have seen no signs of increased thyrotoxicity during the action of radioiodine in 30 patients and this disturbance was only reported as evident when an iodine decaying more rapidly than I^{131} was being used in treatment.

The last theoretical objection is the possible induction of carcinoma many years later. This possibility has been widely discussed and I have little to add. It is known that intense irradiation can be carcinogenic although the examples occurring in man have usually, but not always, involved long-

continued irradiation: by radium in bone, or radon in lungs, by X-rays to the skin and possibly by thorium in the liver or bones. The thyroid irradiation has a short mean duration of a few weeks and may prove to have no late carcinogenic effect. Certainly no such results have been reported from its use, but even the earliest treatments were given only ten years ago and a latency of twenty years would not be unlikely. Similarly, the X-ray therapy of Graves's disease does not appear to have caused thyroid carcinomata although this irradiation will have been less intense than may occur locally in the gland during radioiodine therapy.

The question at issue, however, is not whether radioiodine therapy may be carcinogenic, but with what frequency it is carcinogenic. If this frequency proves to be over 1%, the treatment will rarely be justified unless both surgery and thiouracil are clearly dangerous. But if the frequency is less than 0·1%, the treatment will be safer than either alternative and should be given. It is clear that indirect evidence, for example from other species, cannot give the exact quantitative answer required, and for which we can only wait for ten more years until the cases first treated have been followed for the necessary period.

The last objection, which is of a more practical nature, is that of predicting a dose which will cure hyperthyroidism without producing myxœdema. There are two main possibilities—of giving the dose that will probably be necessary, and of giving repeated smaller doses until the necessary effect has been obtained. In the first method the calculation proceeds on the basis that the roentgen dose to the thyroid, per millicurie given, depends on the percentage that will be taken up by the thyroid, and on the mass of thyroid tissue through which it will be spread, as well as on the time during which it stays in the thyroid. These values can be found by examining the uptake and discharge of a test dose, although the test and therapy doses may not be metabolized identically, so that another unknown arises here. The percentage uptake is usually estimated but matters least, as it differs relatively little in different cases. The gland size is awkward to determine accurately and the discharge rate is easily determined at the expense of a few days' delay. The full analysis is not a major investigation and would be only moderately irksome in routine clinical use. It seems possible, however, that individual differences in the radiosensitivity might still be sufficient to cause myxœdema in one patient at a roentgen dosage to the gland which was insufficient to cure hyperthyroidism in another. In several long series of patients so treated, however, the frequency of permanent myxœdema has been kept below 10%, although an average of only 1·3 doses per patient has been needed to cure the hyperthyroidism.

The alternative method of repeated smaller doses has its own difficulties in that the full effect of one dose of I^{131} may not be clear for a number of weeks afterwards, so that control of symptoms may be unduly or dangerously slow. The need here is for a test which can be made within a few days of a dose and which will forecast the full effects that the dose will ultimately have, and this problem has not yet been solved, although I think it is soluble. The use of a radioiodine with a short half-life would probably make both the test and its interpretation easier, but would increase the whole body irradiation associated with adequate therapy.

At present, therefore, the radioiodine therapy of Graves's disease is somewhat unpredictable as to dosage and clumsy to the extent that, although hyperthyroidism can always be cured, a permanent myxœdema will sometimes follow. My view is that this is not at present the treatment of choice for Graves's disease but might well become so if, in ten years, the anxiety as to carcinogenesis is shown to be unfounded, and if, as seems likely, a scheme of dosage is developed which is efficient in curing thyrotoxicosis without causing myxœdema. Meanwhile I think this treatment is valuable for patients over 45, for whom both surgery and thiouracil are undesirable and for whom the expectation of normal life is comparable with the latency of any possible carcinogenic effect; and in younger subjects if both these alternatives are clearly contra-indicated; and in particular, perhaps, in older patients with mental disturbance and younger subjects with post-operative recurring thyrotoxicosis. It should not be used in pregnancy and probably not in the presence of severe tracheal obstruction, and would not be used if the gland were suspected of possible malignancy.

When we turn to the radioiodine treatment of thyroid carcinoma, the main problem again lies in selecting those cases which are suitable for treatment, rather than in the conduct of treatment in suitable cases. I would in any case wish to discuss selection and not treatment, because we have had no cases under treatment for longer than three years, so can form no estimate of the likelihood of final cures.

Let me say first that radical excision is, of course, the treatment of choice whenever possible. Of the inoperable tumours, the aim is to select those which concentrate radioiodine sufficiently actively—but any evidence of selective concentration would probably justify an attempt at radioiodine treatment.

If this were all, the problem would be merely technical. It has often been shown, however, that uptake may be revealed after destruction of all normal thyroid tissue in a tumour in which no uptake was detectable before thyroid ablation (using the word ablation to cover total thyroidectomy as well as radioiodine destruction of the thyroid). In some cases, the thyroid ablation may simply render it technically easier to detect the radioiodine uptake in the tumour. The tumour uptake in other cases has been proved to have been increased or initiated after thyroid ablation. Whichever mechanism is responsible, the practical consequence is that we must select, not only tumours which can be shown

to concentrate iodine, but those which are likely to do so after thyroid ablation. For this the tumour histology is probably the best criterion, and the presence of abundant colloid correlates reasonably well with the capacity for iodine concentration.

This brings us to the tests for uptake, whether made before or after thyroid ablation. The most obvious is by biopsy a few days after radioiodine, comparing the radioactivity of a given weight of tumour tissue with that of an equal weight of plasma or muscle. This method fails with biopsy material from the neck which might contain normal thyroid tissue, when an autoradiogram will be needed to show that the uptake was actually in tumour tissue. There are several difficulties about this otherwise valuable method. Firstly a large dose of several millicuries is needed if a negative autoradiogram is to indicate that radioiodine treatment would be ineffective. Secondly, many biopsies may be needed if the activities of different metastases, and at different times before and after ablation or during treatment, must be established; and even a single biopsy may be impracticable. Moreover, biopsy gives information only of iodine uptake with retention until the day of the biopsy, and might fail to reveal uptake in a tumour which concentrated iodine well but lacked the mechanisms for its retention.

For these reasons, external counting over the body surface seems to be a more practical, if a more complex, method of diagnosing radioiodine uptake. I do not intend to discuss the details of counter shielding on which depends the efficiency with which uptake in a tumour can be discriminated from that in adjacent structures. Two general methods of shielding are of value, one in which the counter is surrounded by a coaxial cylinder of lead, and is used to map the local distribution of radioiodine under the skin from point to point, the other with shielding parallel to the counter axis, to examine the "profile" of iodine distribution along the length of the body axis.

We are finding it of value to make daily profile measurements after a test dose to distinguish any abnormal site of concentration from those which are normal at a given stage after the dose; and to identify the organ responsible for any peak on this profile distribution by local counting over the relevant area. Iodine is normally concentrated in the saliva, gastric juice, milk and urine while it is in the circulation as iodide, and later in the liver while circulating as thyroxine, as well as in the thyroid; so that many sites of concentration are normal at different stages after the dose.

We have investigated the radioiodine uptake of 25 thyroid tumours which were all, on histological grounds, certainly carcinomatous and consistent with a primary thyroid origin. Of these, 11 were largely or wholly anaplastic, while 14 were differentiated. In the anaplastic group, iodine uptake was probably present in only 2 of the cases examined before thyroid ablation. The thyroid was ablated in 5 cases of this group and uptake was probably present in 2 of these cases tested after ablation. No patient from this group, however, is surviving despite attempted radioiodine treatment in 5 cases.

In the 14 cases with greater histological differentiation the tumour could be shown to take up iodine before thyroid ablation in 2 cases with certainty and probably in 5 further cases. In the 6 cases within this group that have been examined after ablation, radioiodine uptake could be demonstrated unequivocally in the tumour in 4 and probably in the remaining 2. Uptake became evident only after ablation in 2 cases in which it could not previously be established.

It may be concluded, therefore, that treatment is at present unlikely to be of value for anaplastic thyroid tumours. The more differentiated carcinomata should certainly be examined for iodine uptake after thyroid ablation since a therapeutically useful degree of such uptake appears only to be demonstrable under these circumstances.

If we try to define the procedure when thyroid carcinoma is suspected, I would suggest the following plan:

Firstly, biopsy.

Secondly, radical excision, if possible. If attempted and found to be impossible, and biopsy has shown a differentiated tumour, the excision should include as much normal thyroid tissue as practicable.

Inoperable tumours that are undifferentiated and appear likely to be radiosensitive should be treated by radiotherapy if sufficiently localized and unless they are shown to take up radioiodine.

With inoperable, but well-differentiated tumours, and certainly for all cases with colloid-filled follicles throughout the tumour, the thyroid should be ablated by total thyroidectomy if possible and otherwise by radioiodine.

After thyroid ablation, radioiodine treatment should be instituted if uptake can be detected or induced in the tumour, or, more simply, if profile counting reveals any abnormal site of radioiodine retention; and continued until no such site remains, if this can be achieved without the development of radiation anæmia.

It will be observed that much of the selection of cases can be done without the use of radioiodine tests, which are rarely profitable or essential before thyroid ablation. For most patients, therefore, no information is lost and valuable time may be gained if a biopsy is performed on the presumptive diagnosis of thyroid carcinoma, and is followed by total thyroidectomy if the tumour proves to be highly differentiated. Radioiodine is only then required after ablation, or for the ablation if thyroidectomy is impracticable.

After mentioning various other points Dr. Pochin concluded by discussing the hazards involved in handling therapeutic amounts of radioiodine, and the result of tests of the efficacy of nursing and laboratory precautions against the various hazards.

Professor E. J. Wayne (Dept. of Pharmacology and Therapeutics, The University, Sheffield):

The Treatment of Thyrotoxicosis with Radioactive Iodine.

Based on work carried out in Sheffield by Dr. A. G. Macgregor and Professor E. J. Wayne in the Department of Pharmacology and Therapeutics of the University, and by Mr. G. W. Blomfield, Dr. H. Miller and Mr. J. C. Jones of the Sheffield National Centre for Radiotherapy.

There are few medical conditions for which we have available, as we have here, three effective forms of therapy—operation, the antithyroid drugs and radioiodine. I shall review the merits and defects of the first two of these methods so that we may consider the type of case in which the third is useful. Although *partial thyroidectomy* is now very safe when carried out by an experienced surgeon and after preliminary treatment with the antithyroid drugs, it may still have a significant mortality, and complications are by no means uncommon. These tend to be less stressed than they should be since surgeons rarely follow up their cases as fully as physicians and the results obtained by surgeons in some large American clinics are by no means completely satisfactory. Quite apart from the risk of damage to adjacent structures the surgeon often takes away either too much or too little of the gland. Thus surgeons who report a high incidence of myxœdema give a low figure for recurrence of the thyrotoxicosis and vice versa (Vander Laan and Swenson, 1947).

The antithyroid drugs are not without risk although in our view their danger has been exaggerated. In some patients the onset of drug fever and rashes limits their use, others are resistant and the long-term results are often disappointing. Goodwin and Wilson (1950) in our clinic found a relapse rate of 63% after withdrawal of the drug and Dunlop and Rolland (1950) have reported a similar experience. It seems as if these substances relieve the patient of his symptoms while he cures himself and if this does not happen he reverts to the thyrotoxic state when the drug is withdrawn.

There would thus seem to be a definite place for a third form of therapy provided it can be shown to be safe and effective.

We believe that radioiodine fills this place and we have now treated over 80 patients with it. We aim to deliver to the gland a dose of about 8,000 to 10,000 roentgens. The *factors governing the actual dose* which the gland receives are (1) the number of millicuries in the drink, (2) the fraction of this taken up by the gland, (3) the rate of elimination of iodine from the gland, (4) the size of the gland. In addition there may be a patchy distribution of the iodine in the gland and variable sensitivity of the tissues to radiation. In spite of our inability to allow for all these factors in any one case, we still feel that an arbitrary fixed dose is less satisfactory than one based on an attempt to eliminate some of the variables. We therefore do not usually give an antithyroid drug before radioiodine therapy unless the patient is very thyrotoxic and we determine with a tracer dose the uptake and total half-life of the radioiodine in the gland. We attempt to assess the size of the gland by making a map using a highly collimated counter in two planes at right angles (Blomfield, Jones, Macgregor, Miller and Wayne, 1951*a* and *b*). Retrospective estimations of the dosage after the therapeutic drink are made and give a measure of the actual amount of radiation received by the gland as a whole. By this method we now aim at producing a complete remission with a single dose delivering 8,000 to 10,000 roentgens to the gland. The technique of giving multiple small doses has a bad psychological effect on some patients and those who use them have to contend with the modifying effect of previous doses. Of 50 patients rendered euthyroid by our technique only 10 have required more than one dose and in our experience myxœdema is just as liable to develop after the second dose as after the first. Nevertheless, we agree that there is a great variation in the sensitivity to radiation of different glands and some patients have become euthyroid with as low a dose as 4,800 equivalent roentgens while others have required a second dose after an initial estimated dose of 10,000 e.r.

The response to treatment is best *assessed* by the clinical features of the case just as with other forms of therapy. We specially note shrinkage in the size of the gland. If there is no clinical improvement three months after a therapeutic dose we then carry out a diagnostic test with radioactive iodine and consider giving a second dose. In the diagnosis of hyperthyroidism we now put the greatest reliance on an estimate of the level of the protein-bound radioiodine in the plasma forty-eight hours after a tracer dose has been given although gland uptake figures are also valuable (Goodwin, Macgregor, Miller and Wayne, 1951). When patients have been subjected either to operation or radioiodine treatment, however, the plasma protein-bound radioiodine correlates less well with the clinical state of the patient than does the peak uptake. We find a significant fall in peak uptake even in those who have not responded well to an initial therapeutic drink and a striking fall in those rendered euthyroid. The fall in protein-bound radioiodine is not as great as one would have anticipated and the levels remain higher than in normal non-toxic individuals.

The results of radioiodine treatment in 80 patients are summarized in Fig. 1. All cases followed for a year or more have become euthyroid or hypothyroid. Little change occurs for a few weeks but objective improvement is usually noticed in about a month. If the patient responds rapidly it is our impression that he will subsequently develop some degree of myxœdema although this may be transient. Normal thyroid function is accompanied by striking gain in weight, by diminution in size of the gland

and by disappearance of the lid retraction. Observations by orbitonometry have shown less tendency for the exophthalmos to increase than when patients are treated by surgery or antithyroid drugs and in some cases it may diminish to a most remarkable extent.

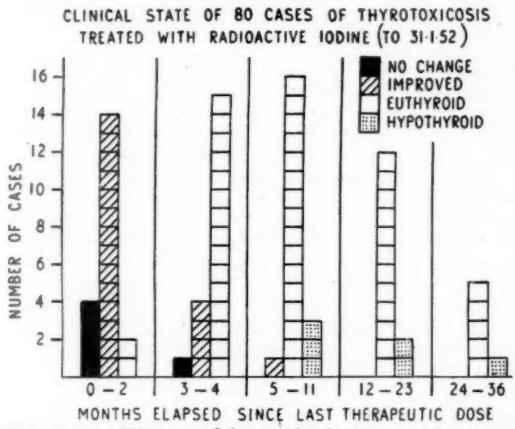

FIG. 1.—Clinical state of 80 cases of thyrotoxicosis treated with radioactive iodine.

We have encountered no immediate complications except in one patient who had an increase in the severity of her thyrotoxic state five days after the therapeutic drink.

Selection of Cases for Radioiodine Treatment

From the patient's point of view this form of treatment is, of course, ideal. A drink of an almost tasteless medicine gives a complete cure. The only hazard is the danger that thyroid carcinoma may develop many years after the course of therapy. This is as yet a hypothetical danger but radioiodine is nevertheless not our first choice in the treatment of young patients. We have, however, no hesitation in using it in those with an expectation of life of less than twenty years. It is therefore always suitable for patients over 50 or for so-called thyrocardiacs. It is the best treatment at any age where thyrotoxicosis has recurred after operation. We also use it in those who have relapsed after methylthiouracil therapy and who are either bad operative risks or who refuse operation. Pregnancy is an absolute contra-indication. We prefer operation in many cases of toxic nodular goitre unless the patient is a poor operative risk, but such patients respond well to this form of therapy. In this group repeated small doses may be preferable since the high doses usually required subject the body tissues to a rather large dose of radiation.

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Professor D. W. Smithers (Director, Radiotherapy Department, Royal Cancer Hospital, London): *The Treatment of Cancer of the Thyroid with Radioactive Iodine.*

About 300 people die from cancer of the thyroid in England and Wales each year so it is not a common disease compared with cancer of the breast with nearly 8,000 deaths per annum, or cancer of the lung with probably more than 11,000. It is only a small proportion of the patients in this comparatively small group with malignant thyroid tumours who benefit from treatment with radioactive iodine. The effect so far produced by the introduction of this method of treatment on the general problem of the management of patients with cancer may reasonably be regarded as insignificant, but it has an interest, out of all proportion to its success, as a new step forward in cancer treatment which allows us at times to achieve something which was never possible before. Those few patients with metastases, now alive and well, who would have died without it are not likely to accuse us of exaggerating its importance.

On the surface at least the position of radioactive iodine in thyroid cancer is unfortunately all too simple; the nearer the tumour approaches in structure and function to the normal thyroid, that is the less malignant it tends to be—that is, the less the need for a new method of treatment—the more likely is the new method of treatment to be effective. In those dispassionate moments when we allow our thoughts to wander from the human problems associated with malignant disease the fine variety of behaviour of thyroid tumours must at least command the respect of those of us who appreciate the versatility of nature. We can even indulge in some sly enjoyment at the knots into which they can tie the tidy minds of the inveterate classifiers and definers amongst our colleagues: the "benign" tumours which show "malignant" invasion of blood vessels, the "benign" tumours which metastasize freely to lungs and bones without doing the patient much harm for twenty years or more, the "malignant" tumours which hastily metastasize to lymph nodes even before they can be detected clinically but whose metastases then show little inclination to metastasize, even when repeatedly attacked by enthusiastic surgeons and radiotherapists. With tumours displaying such bold disregard for the rules, and with the possibility of many years of life without treatment for some of these patients, despite disseminated metastases, we must be particularly careful in judging any treatment method. Radioactive iodine is not needed for the less malignant localized tumours and is ineffective for the more malignant, undifferentiated, disseminating tumours. Its present value lies in the treatment of a few patients with well-differentiated tumours which have either advanced locally to the point of inoperability through neglect or mismanagement or have metastasized. Its advance in the future lies in our ability to increase the uptake of iodine in the tumours in this group.

In the past three years at the Royal Cancer Hospital we have investigated the uptake of I^{131} in 29 patients with thyroid cancer and been able to use it therapeutically in 8. At the Christie Hospital and Holt Radium Institute in Manchester they have tested the uptake in 43 patients but found only 5 suitable for treatment. All these 5 had highly differentiated adenocarcinomas with colloid formation, but 6 others with a similar histological picture failed to take up iodine sufficiently for treatment. 3 of their patients are alive and well. The first, with multiple bone deposits, was a woman of 63 treated in August 1949; the second, a man of 61 with multiple lung deposits, was treated in April 1950; and the third, a woman of 31 also with multiple deposits in the lungs, was treated in June 1950 (Paterson, 1952). A few such patients are now to be found in most of the larger treatment centres.

Interest at present centres in attempts to find methods of increasing the uptake of radioactive iodine in thyroid cancer. The first and obvious method was to remove all normal functioning thyroid tissue. This was done either by total surgical thyroidectomy or by the administration of radioactive iodine used to destroy the normal thyroid first in the hope that it might be used again to destroy the tumour later. Some success with this method was reported but nearly always in patients whose tumours were already taking up iodine fairly well, but in whom matters were improved by removing the competition of the normal gland. In Manchester they tried this method of thyroid elimination in 16 patients with non-functioning thyroid cancer without obtaining any improvement in iodine uptake in a single one.

The other methods that have been tried to increase uptake are the administration of thyrotrophic hormone, which has not proved to be very helpful, or of thiouracil, which has. In those patients in whom thiouracil has been used a thyroidectomy has been performed first so that it is not always easy to say whether increased uptake is due to a late effect of thyroidectomy alone or a direct effect of the thiouracil. Thiouracil has been given in doses of from 1 to 1·5 grammes a day following total thyroidectomy and every six weeks the administration of thiouracil stopped for forty-eight hours before giving a tracer dose of radioiodine. When the tracer study has been completed the thiouracil treatment is continued if no increased uptake has been shown, and the process repeated either until some uptake is found, until the patient becomes too ill to continue, or until the patience of the investigators is exhausted. Rall, Miller, Foster, Peacock and Rawson (1951) report the induction of uptake of radioactive iodine in 21 out of 35 metastatic cancers of the thyroid using a combination of total thyroidectomy and prolonged thiouracil treatment.

There have been three explanations put forward to account for the effect of thiouracil in increasing iodine uptake in carcinomas. The one which was responsible for its use in the first place was that as the result of having taken thiouracil for a long period of time the patient is reduced to a state of iodine depletion so that the avidity for iodine of any tissue remotely capable of functioning as thyroid becomes extremely high. The second explanation put forward is that thiouracil by causing myxœdema stimulates the pituitary to put out an increased amount of thyroid stimulating hormone which then persuades the cancerous tissue to function. The third explanation is that large doses of thiouracil augment the action of the patient's own circulating thyroid stimulating hormone.

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Section of Otology

President—G. E. ARCHER, M.B., Ch.B., F.R.C.S.Ed., D.L.O.

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The Pathology, Symptomatology and Diagnosis of Certain Common Disorders of the Vestibular System

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INTRODUCTION

IT is generally agreed that within the last fifteen years some extension has been achieved of our understanding of the problems of human vertigo. Nevertheless, difficulties and confusion still persist and in the course of the present communication an attempt will be made to advance the process of its clarification, both by critical review and by a short account of some of our recent investigations. The foundations of the subject are to be found in the writings of Prosper Ménière, and these we take as our starting point.

Ménière's papers on vertigo are chiefly remarkable for the powers which their writer displays of describing and analysing the symptoms and signs of disease. It is easy now to realize that it was this mastery of symptomatology which, more than anything else, enabled him to identify, with an accuracy which has never been seriously questioned, the disease of the labyrinth which has since come to bear his name. Beyond, however, asserting with confidence that the disease was due to a lesion of the internal ear, limited to that organ and indestructive to life, he made no *direct* observations upon its pathological anatomy, and further information upon this point was not forthcoming until 1938. Since then the morbid anatomical basis of the disease has been established by means of histological examinations of the temporal bones in a number of clinically characteristic cases. It is fair to say that this opinion is not perhaps a universal one. On the contrary, it is still sometimes said that Ménière *did* describe the pathological anatomy of Ménière's disease, and reference is made to the case which Ménière cited of the young girl who died after a short illness, due to a chill, in the course of which she suffered from deafness, vertigo and vomiting. Ménière examined the temporal bones and found a reddish exudate in the semicircular canals. But no reader of his text could suppose that Ménière wished it to be inferred from this observation that the cause of *this* girl's illness was the same as that responsible for the recurrent attacks of vertigo with deafness so characteristic of the other, but essentially benign, disease which he had described so clearly. Indeed, the fatal issue of the case alone rules out this possibility.

Our own view is that Ménière's reference to the anatomical changes in these labyrinths had an entirely different significance and one, moreover, which is quite obvious from the context.

It is necessary to bear in mind that in 1861, when Ménière published his best-known paper, the very possibility that a lesion of the internal ear could cause such severe symptoms as vertigo and vomiting was still a moot point.

Flourens' experimental work on pigeons, in which he demonstrated for the first time that gross disorders of equilibrium could be produced by injury of the semicircular canals, had only been published some thirty years earlier, and its significance as regards the problems of human disease had not yet been appreciated. It was Ménière's great merit that he knew of Flourens' work and understood its meaning. To him, therefore, the significance of the exudate in the semicircular canals of his patient was that in it he recognized the vital link so far missing between Flourens' animal experiments and the problem of human vertigo, a demonstrable lesion of the semicircular canals in a subject who had suffered in life from vertigo. Ménière argued that if such a hæmorrhagic lesion, occurring in the course of this girl's fatal illness, could produce vertigo, then other lesions of the inner ear, be their precise nature what you will, could also be the cause of the vertigo in his other group of subjects whose symptoms he had described with such care. There Ménière left the matter. He had identified his disease by his accurate specification of its symptomatology and natural history. As to its pathology he said only this: " La lésion matérielle réside dans les canaux semicirculaires"—"the essential lesion is situated in the semicircular canals". Little more knowledge came our way until 1938 when Cairns and Hallpike examined the temporal bones of their two subjects and established the nature of Ménière's lesion [1].

Of this the essential abnormality was a distension without evidence of infection or trauma of the endolymphatic vesicle; a finding, remarkable in itself and since reproduced with remarkable uniformity in a considerable number of further histological studies of the temporal bones carried out by Hallpike and also by a number of others.

To summarize this chapter of history it can be said that Ménière's concept was of a disease *sui generis* of the internal ear. This he based in the main upon his own studies of symptomatology and supported it by

certain *in*direct evidence of animal expermentiation and of human pathology. Since 1938 this concept has been abundantly confirmed by further clinical studies and by *direct* pathological evidence derived from the study of human temporal bones.

This analysis of Ménière's work and the position it occupies in the much larger field of organic vertigo as we know it today was clearly stated by Cairns and Hallpike in their original paper, and has since gained wide acceptance by a number of American authors, in particular Day [2], Wells [3] and Williams [4]. But since Ménière's time this field has become much enlarged and its contours very confused and it has been customary to mix together with Ménière's disease, under the undiscriminating label of Ménière's syndrome or even of pseudo-Ménière's disease, other types of organic vertigo which conform only vaguely to the established symptomatology and pathology of Ménière's disease. In the past this has been unavoidable, and connotes only our ignorance of the clinical and pathological features of these disorders which would enable us to distinguish them from each other and from Ménière's disease.

It is, of course, the task of oto-neurological research to resolve this ignorance. When complete, it will be possible so to distinguish them and call them by names which will announce their clinical and pathological individualities. When that time comes the need for the label, Ménière's syndrome, will disappear and no more will be heard of that lexicographical abomination, "pseudo-Ménière's disease". The task has been begun with the clinico-pathological work carried out during the last fifteen years upon Ménière's disease. During this, we have seen rescued from the confusion what Ménière put into it—his disease—with its symptomatology confirmed and its pathology established. It is the purpose of the present communication to present certain new information upon the subject of Ménière's disease. In addition, descriptions will be given of two other varieties of organic vertigo; one a disorder of the vestibular neurones; another a disorder of the otolith system in the labyrinth. Both of these are liable to be confused with Ménière's disease, and carry Ménière's name on that familiar label "Ménière's Syndrome". It will, however, be shown that their clinical and pathological features make obvious their distinction from Ménière's disease, and other labels will accordingly be proposed for them which declare this distinction and are more appropriate to their symptoms, signs and pathological anatomy.

In the present work we have endeavoured first and foremost to make use of the clinical methods in which Ménière himself so excelled. That is to say, it has been our aim to base our opinions primarily upon the study of symptoms and natural history of disease. To this we have added as much evidence as possible from the physical signs which can now be derived from modern tests of cochlear and vestibular function. Finally, when the opportunity has presented itself we have been able to check our clinical evidence by histological studies of the temporal bones.

Part I.—Ménière's Disease

On symptomatology there is little to add to Ménière's original description and to the analysis published in 1942 by Cawthorne *et al.* [5]. We make more, perhaps, of distortion of hearing than Ménière did, and of the exacerbation of tinnitus and deafness during the attacks. But these can hardly be described as major developments. As regards physical signs we find the vestibular test results are still very much as previously stated [5]. In particular the caloric test results are abnormal in 94% of cases. Of these, 20% show a directional preponderance towards the sound ear. In 58% there is a loss of canal sensitivity in the affected ear. In 16% the result represents a mixture of these two primary derangements. In the field of tests of cochlear function, however, we are able to report considerable progress since 1942. Dix *et al.* [6] showed in 1948 that the phenomenon of loudness recruitment was invariably present in Ménière's disease, and that in a proportion of such subjects over-recruitment made its appearance. In another series of observations Hood [7] has been able to show that the phenomenon of adaptation is abnormally rapid in cases of Ménière's disease. Speech audiometry, too, has in many cases given results in cases of Ménière's disease which have a high diagnostic significance. In particular they reveal a loss of intelligibility which is out of proportion to the pure tone audiometric threshold loss. It will be recalled that in the publication to which reference is made [6], the characteristically positive finding of recruitment in Ménière's disease was compared with its characteristic absence in cases of VIII nerve tumour, and thereon was based our opinion that the phenomenon of recruitment was attributable to hair-cell disease. For this argument pathological evidence is in some cases very strong. That is to say, the cells of Corti's organ show very striking changes while the cochlear nerve fibres and the cells of the spiral ganglion are quite normal. This is exemplified in the following photomicrographs of the organ of Corti in the normal and affected ears in the first of the two cases of Ménière's disease described (Figs. 1 and 2). [1]. (For Figs. 1 to 6 *see* pp. 347 and 348.)

In other cases, however, examined both by ourselves and others, these changes in the hair cells are by no means obvious, and to circumvent the difficulty it has been necessary to have recourse to the argument that in such cases the histological changes responsible for the deafness and recruitment were of a kind which made it possible for them to be masked by the considerable histologic artefacts which are so often unavoidable with human material. If this argument be accepted, then with it must also be accepted the likelihood that in a case of unilateral Ménière's disease, in which this histological artefact was reduced by exceptionally favourable conditions of fixation, it should be possible to distinguish some significant anatomical changes in the hair cells. A brief account of clinical and pathological data derived from such a case now follows:

The case was that of a man of 54 who died in 1949. In 1938 he began to suffer from attacks of paroxysmal vertigo, during which objects rotated vertically. They were accompanied by nausea and

lasted from ten to thirty minutes. Consciousness was not lost during the attacks, during which he was compelled to lie down. He had experienced such attacks yearly until 1945. During that time he also had buzzing tinnitus in the right ear and deafness which was progressive. He was seen at Sir Charles Symonds' Out-Patients in November 1948 on account of epileptic attacks lasting for half an hour or more, during which he became unconscious. Soon after, he developed drowsiness, confusion and a severe affection of speech. He was admitted to Queen Square on February 9, 1949, where a diagnosis was made of a left temporal lobe neoplasm. This was confirmed by biopsy; the patient died six days later. At post-mortem a large tumour was found in the left temporal lobe which histological examination showed to be an astrocytoma. Apart from some herniation of the inner edge of the left temporal lobe through the incisura tentorii on the left-hand side and some cerebellar coning, there was no other abnormality of the cerebrum, cerebellum, brain-stem, cranial nerves or meninges. The last otological examination was carried out at Guy's in December 1948 when the findings confirmed the diagnosis of Ménière's disease, the nose and throat being found healthy, the tympanic membranes normal with a severe degree of deafness of the right ear of the perceptive type. Pure tone audiometry showed an approximately uniform hearing loss on the right side of some 60 to 70 decibels. There was, in addition, a slight high-tone loss in the left ear confined to the frequencies 4,000 and 8,000 cycles. Caloric responses showed a slight reduction of the responses to both cold and hot stimuli on the right-hand side. We were able to obtain the temporal bones sixteen hours after death, and to undertake their fixation and preparation ourselves. It was possible to display the histology of the labyrinths in a comparatively good state of preservation; in particular, the condition of the hair cells was sufficiently free from post-mortem artefact to make worth while a close morphological comparison of Corti's organ in the two ears.

In Fig. 3 is shown the unaffected cochlea with a normal spiral ganglion and a well-preserved organ of Corti with Reissner's membrane in its normal position.

In Fig. 4 is shown the affected cochlea; the spiral ganglion is of normal density. The general structure of Corti's organ can be made out, although not in any great detail. Finally, the typical distension of the scala media is seen with the displacement of Reissner's membrane.

To facilitate comparison of the structure of Corti's organ in these two labyrinths, photomicrographs have been prepared in which views of the organ in the different coils of the unaffected cochlea are presented at high magnification side by side with their counterparts in the affected cochlea.

In Fig. 5 are shown the organs of Corti in the anterior and posterior middle coils. The organs of the right or affected cochlea are on the right-hand side. On the left, the normal side, the normal form and size of Corti's organ is well preserved in both coils. Both rods of Corti are well seen, and the form of Corti's tunnel is nearly normal. In both coils can be seen what is probably the remains of a hair cell and its nucleus. On the affected side obvious changes are present. Thus, the total size of Corti's organ is reduced, its shape is irregular, the outer rod has been demolished and the hair cell framework has disappeared. In addition, Corti's tunnel, or what is left of it, seems to be occupied by a kind of coagulum.

In Fig. 6 are shown the anterior and posterior basal coils. As before, the right or affected organ of Corti is shown on the right. On the left or normal side, Corti's tunnel is preserved and the organ is of normal size and shape. There is distinct evidence of at least one hair cell nucleus, while a nerve filament is to be seen crossing Corti's tunnel. On the affected side, however, the outer rod of Corti has disappeared and some coagulum is present in the space of what was Corti's tunnel. Finally, the area occupied by the hair cells seems to be disorganized, both as regards its shape and its cellular contents.

These changes have been demonstrated in some detail since they strengthen in a very satisfactory manner, what has been a weak link in the chain of argument, which has led us to conclude that the deafness in Ménière's disease and the loudness recruitment phenomenon which is so characteristic of it are attributable to hair-cell disease.

Part II.—Vestibular Neuronitis

We come next to another group of patients whose chief symptom is again vertigo, usually but not always paroxysmal in character. This group is chiefly distinguished from Ménière's disease on clinical grounds by the conspicuous absence of cochlear signs and symptoms. We began to recognize this condition at our Clinic at Queen Square as a distinct clinical entity in about 1946. For a variety of reasons, to which further reference will be made, it seemed then attributable, beyond any doubt, to some form of organic disease confined to the vestibular apparatus and localized, in all probability, to its peripheral nervous pathways up to and including the vestibular nuclei in the brain-stem. It was impossible, however, to go further and specify the particular elements of the neurones, cells or fibres, which were affected. When, therefore, it came to naming the condition we required a term comprehensive enough to encompass this uncertainty. We chose the name "vestibular neuronitis" and have since continued to use it.

We first described the condition in 1949 in a short communication to the Fourth International Congress of Oto-laryngology in London [8] and our present analysis of its clinical features is based

upon the study, made possible through the courtesy of our colleagues at Queen Square, of over 100 cases. These we have examined in the course of the last few years. Age and sex distribution are tabulated as follows:

TABLE I

Age	*Age Distribution*					Over		*Sex Distribution*			
(years)	Under 20	20–29	30–39	40–49	50–59	60	Total	Male	57		
Number ..	5	21	37	22	14	1	100	Female	43	Total	100

It will be seen that the disorder chiefly affects the age group 30 to 50 without preference for sex. Apart from the absence of cochlear signs and symptoms the condition is often but not always distinguishable from Ménière's disease by the character of the vertigo. This may consist of sudden and transient seizures accompanied by sensations of blackout. On the other hand there may be no severe paroxysms and the disequilibrium may take the form of "feeling top heavy" or "off-balance", particularly when walking or standing. As in other forms of organic vestibular disease, the disequilibrium is aggravated by head movements of all kinds. In a fairly high proportion of the subjects the onset of the symptoms is associated with some kind of febrile illness, or with evidence of infection of the ears, nose and throat, and to this aspect of the malady we shall later return.

Otoscopic findings are typically normal with normal test results of cochlear function including pure tone audiometry.

When we come to investigate vestibular function, however, very marked abnormalities are always present, in particular of the caloric responses which are consistently reduced, often grossly so, and on both sides. A full description of the caloric abnormalities follows: All tests were carried out in accordance with the technique described by Fitzgerald and Hallpike [9]. We have divided our test results in accordance with the following nomenclature:

(1) *Complete canal paresis.*—No response obtained with irrigation at 20° C. for one minute.

(2) *Severe canal paresis.*—No response obtained with stimuli of normal strength, i.e. 30° C. or 44° C. for 40 seconds; a response, however, was obtained with water at 20° C. for one minute.

(3) *Moderate canal paresis.*—In this group responses, although obviously reduced, were obtained with stimuli of normal strength.

Certain of the cases exhibited directional preponderance, and others a combination of directional preponderance and canal paresis. For further particulars of the qualitative and quantitative assessment of abnormalities of the patterns of the caloric test results reference should be made to previous publications [5, 10].

The abnormalities were further divided into two main groups:

(1) Bilateral. (2) Unilateral.

The complete classification is given in Table II.

TABLE II.—CALORIC ABNORMALITIES

Bilateral:	Complete canal paresis ..	7	*Unilateral:*	Complete canal paresis ..	8
	Severe canal paresis ..	25		Severe canal paresis ..	9
	Moderate canal paresis ..	7		Moderate canal paresis ..	20
	Combined canal paresis with directional preponderance to the same side	8		Combined canal paresis with directional preponderance to the opposite side	8
	Total	47		Directional preponderance to the opposite side ..	8
				Total	53

It will be seen from Table II that substantial abnormalities of the caloric responses were present in all of our 100 cases. In 47 they were bilateral; in 53 they were unilateral.

In a few of the cases the abnormalities of the caloric test findings have been shown by follow-up to be the first and only manifestation of disseminated sclerosis. In the great majority, however, there was no evidence whatsoever of extra-vestibular nervous disease, and in these we have postulated an organic lesion of the vestibular nervous pathways at some point up to and including the vestibular nuclei, a vestibular neuronitis. We have preferred to believe that the lesion is central to the labyrinth in accordance with a well-established principle of oto-neurology, namely that destructive labyrinthine lesions, whatever their pathology, tend on the whole to involve the cochlear apparatus. Furthermore, a high proportion of our subjects have been found to exhibit significant changes of the galvanic test responses, evidence which is strongly indicative of a lesion central to Scarpa's ganglion. Later reference will be made to the technique and interpretation of these tests.

The condition is essentially a benign one. It responds well to treatment of focal infection when this is present, and generally recovers in the course of a few years. In a few cases we have observed the re-establishment of the caloric responses.

We propose now to consider in greater detail two important clinical aspects of the condition:

(1) The role of infection as a pathogenic factor.

(2) The galvanic test results and their localizing value.

(1) *The role of infection as a pathogenic factor.*—The role of focal infection as a cause of organic vertigo has been discussed by a number of authors, notably by A. J. Wright [11] and particular attention was directed to this point by Cawthorne *et al.* [5]. The evidence adduced therein showed that in subjects in whom the diagnosis of Ménière's disease had been made upon the basis of an adequate analysis of symptomatology and physical signs, infective foci in the nose and throat occurred so rarely as to make it difficult to attach thereto any causal significance. In vestibular neuronitis, however, our clinical investigations have led us to the conclusion that infective foci in the nose and throat play an important part in its pathogenesis. This view is based chiefly upon a study of 50 of our cases in whom, in addition to a routine examination of the nose and throat, we have also carried out blood examinations including sedimentation rates and radiological examination of the paranasal sinuses. The sedimentation rate tests were carried out according to the Westergren technique. We have regarded as pathological values for the first hour which exceed 8 mm. in the male or 10 mm. in the female.

Our 50 cases fall into the following three main divisions with certain subgroups:

DIVISION I.—CASES PRESENTING DIRECT EVIDENCE OF AN ACTIVE INFECTIVE FOCUS.

Group A. Antral infection confirmed by the presence of pus on proof puncture: Number of cases, 10. In 4 of these the sedimentation rate was raised.

Group B. Sore throats with obvious evidence of active tonsillar infection: Number of cases, 2. In both, the sedimentation rate was raised.

DIVISION II.—CASES PRESENTING STRONG PRESUMPTIVE EVIDENCE OF AN ACTIVE OR QUIESCENT INFECTIVE FOCUS.

Group A. Definite radiological evidence of antral infection: Number of cases, 6. In some, proof puncture was refused; in others it was negative. In none of these was the E.S.R. raised.

Group B. Dental infection: Number of cases, 4. The E.S.R. was raised in one case.

Group C. Cases presenting no evidence of infective focus, but giving a clear history of an infective illness at the time of onset of the vertigo: Number of cases 13. In 3 of these the sedimentation rate was raised.

DIVISION III.—CASES PRESENTING NO EVIDENCE OF AN INFECTIVE FOCUS, OR A HISTORY OF INFECTIVE ILLNESS.

Number of cases, 15. In 4 of these the sedimentation rate was raised.

Our opinion, based upon these findings, that infective processes play an important part in the pathogenesis of vestibular neuronitis, is chiefly derived from the two groups of cases comprised in Division I. From these we see that clear evidence of antral or tonsillar infection was present in 24% of our 50 cases. This far exceeds any corresponding figures that we could base upon our experience of Ménière's disease. The same can be said of Division II, from which we see that 46% of our 50 cases either gave a clear history of an infective illness at the time of onset of the vertigo, or else exhibited significant evidence of an infective focus in the nose and throat.

In the other 50 of our 100 cases our examinations for evidence of focal infection have been less complete; that is to say, systematic sinus X-rays and blood examinations have not been carried out. Nevertheless, in these remaining 50 cases we have observed clear evidence of antral infection in 8, and in 14 others there was history of an infective illness at the time of the onset of the symptoms.

(2) *The galvanic test results and their localizing value.*—The use of the galvanic tests in vestibular neuronitis would appear to be particularly appropriate since the work of Huizinga [12], Dohlman [13] and others has suggested that the galvanic responses depend upon the integrity of Scarpa's ganglion and the vestibular neurones central thereto, and are preserved in lesions of the peripheral sense organs. We have, therefore, carried out systematic galvanic tests in a number of our patients [14]. Details of the test procedure are given as follows:

"Bipolar stimulation was used, the current being passed between brine-soaked pads firmly located upon one or other of the mastoid processes and the manubrium sterni. The tests were carried out with the patient standing with eyes closed and the feet and heels close together. The reaction was described as positive when swaying, which normally occurs towards the ear carrying the positive electrode (anode), could be clearly and repeatedly observed. Corresponding observations were also carried out with the polarity reversed; in these circumstances, of course, the direction of the swaying was also reversed. . . .

"To begin with, a series of control observations was carried out upon 12 normal individuals with no history of ear disease or vertigo. In all of these it was found possible to observe positive responses with values of current lying within the range of 0·3 to 1·9 mA., with an average value of 0·8 mA."

Full details of these findings are given in Table III, while the values obtained in 16 cases of vestibular neuronitis are given in Table IV. In both tables the figures given are for milliamperes of current at threshold. The maximum currents given in Table IV were determined by the onset of the usual painful sensations from the skin areas underlying the electrodes.

TABLE III.—12 NORMAL SUBJECTS

Ear Stimulated

	Right		Left	
	Polarity		Polarity	
Case No.	−ve	+ve	−ve	+ve
1	0·6	0·3	0·6	0·6
2	0·5	0·45	0·5	0·45
3	0·5	0·5	0·4	0·6
4	0·5	0·7	0·5	0·7
5	1·0	1·0	0·5	1·2
6	0·45	0·4	0·45	0·4
7	1·4	1·5	1·4	1·5
8	1·0	1·3	1·0	1·4
9	0·5	0·7	0·5	0·7
10	0·45	0·4	0·45	0·4
11	0·6	0·6	0·55	0·55
12	1·8	1·9	1·7	1·8

(Table III is quoted from *Brain*, 1949, **72**, 243.)

TABLE IV.—16 CASES OF VESTIBULAR NEURONITIS

Ear Stimulated

	Right			Left		
Case No.	Polarity		Max. current used	Polarity		Max. current used
	−ve	+ve		−ve	+ve	
1	Absent	3·0	4·0	1·3	1·2	
2	Absent	Absent	5·0	Absent	Absent	5·0
3	1·4	1·4		0·6	0·6	
4	0·7	0·7		3·0	3·0	
5	1·6	1·6		1·4	1·4	
6	2·0	3·0		3·0	3·0	
7	Absent	Absent	3·0	Absent	Absent	3·0
8	1·2	1·5		1·2	1·5	
9	1·0	3·0		4·0	1·0	
10	Absent	Absent	5·0	Absent	Absent	5·0
11	Absent	1·5	3·0	1·5	Absent	3·0
12	0·5	1·0		0·7	0·6	
13	Absent	Absent	2·2	Absent	Absent	2·2
14	1·0	4·0		5·0	5·0	
15	Absent	Absent	5·0	Absent	Absent	5·0
16	0·6	0·6		1·0	1·0	

It will be seen that in all, except 3 cases, a significant reduction of the galvanic responses was present, a finding which is certainly suggestive of a lesion of the vestibular neurones involving either Scarpa's ganglion, or the vestibular neurones central thereto. It must be added, however, that the mechanism of the galvanic responses is in some ways obscure, and we have observed their derangement both in cases of severe and long-standing Ménière's disease and in certain other disorders in which it might be supposed that the vestibular end organs are chiefly or solely involved. This finding would be explained upon the reasonable supposition that the response to galvanic stimulation is mediated, at least in part, by the peripheral sense organs. If these are eliminated by disease, then the responses will be reduced but not abolished. The matter is further complicated by the fact that long-continued and heavy sedation is likely in cases of Ménière's disease to reduce the sensitivity of the central vestibular elements. For this reason, too, one would expect, in long-standing cases of Ménière's disease, to encounter some reduction of the galvanic responses. The subject, however, is a difficult one and clearly calls for further examination. So far, however, as we have considered the matter, it would seem clear that the reduction of the galvanic responses in vestibular neuronitis exceeds in degree what we have encountered in Ménière's disease, and therefore supports our hypothesis of a central affection of the vestibular neurones.

FIG. 1.—Organ of Corti. Normal cochlea. The organ is of normal form and size, with good preservation of Corti's tunnel. Details of the hair cells are obscured by post-mortem degeneration. (× 194.)

FIG. 2.—Organ of Corti. Affected cochlea. The organ is shrunken and its outline irregular. Corti's tunnel is occupied by a structureless coagulum. (× 194.)

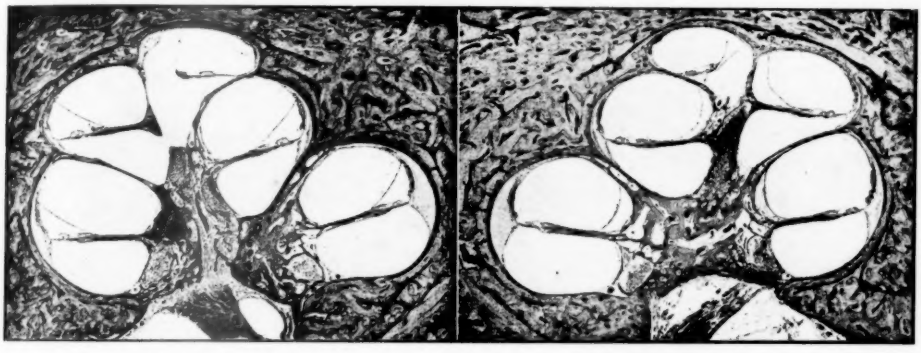
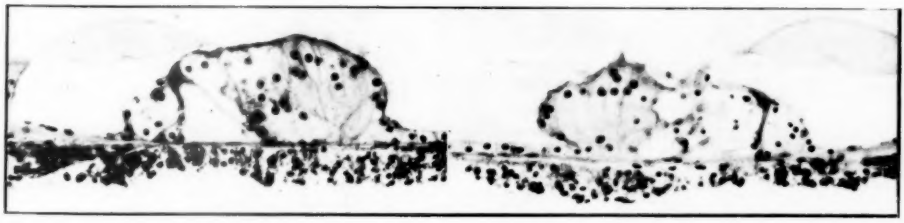


FIG. 3.—(× 10.)

FIG. 4.—(× 10.)

Anterior middle coil.



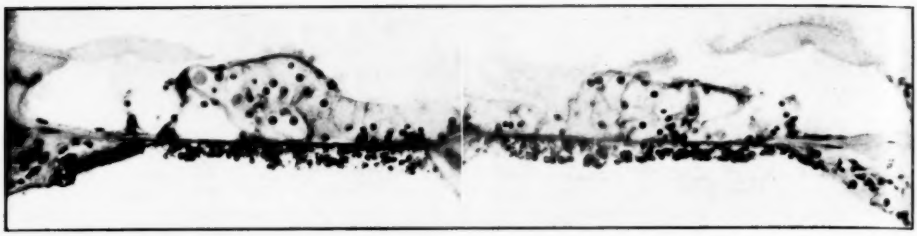
Normal cochlea. Affected cochlea.

Posterior middle coil.

Normal cochlea. Affected cochlea.

FIG. 5.—(× 194.)

Anterior basal coil.



Normal cochlea. Affected cochlea.

Posterior basal coil.

Normal cochlea. Affected cochlea.

FIG. 6.—(× 194.)

We come, finally, to a third variety of organic vertigo presenting as its chief clinical feature a highly distinctive type of positional nystagmus. This again is distinguishable on clinical grounds alike from Ménière's disease and from vestibular neuronitis. Further, we have good reason to believe that its pathological basis is also quite different from those of these two conditions. We shall consider it in some detail.

PART III.—POSITIONAL NYSTAGMUS

This strange and dramatic disorder was first described by R. Bárány in 1921 [15]. Bárány mentions two distinct conditions, one of which he attributed to otolith disease. This we shall describe as Bárány's first type. His second type, to which he referred very cursorily, he seems to have attributed to a lesion within the central nervous system.

Bárány had not a great deal to say about either of these conditions, but what he did say was very much to the point, and in due course we shall return to his own words upon the subject.

Since his time many papers have been written about it, short, long and in many languages. Nylén's clinical and animal studies are well known, in particular his monograph on positional nystagmus occurring in intracranial tumours [16], and in a recent survey of the subject [17] he gives a bibliography of no less than 297 papers written by 192 authors.

Considerable difficulties have been encountered in the preparation of the present communication; difficulties which arise in the main from certain serious deficiencies in the terminology of the subject which now pervades its extensive and confusing literature. Thus, it now seems to be generally agreed that a certain type of positional nystagmus which Nylén defined as the "position changing" type is associated with posterior fossa lesions. This type of positional nystagmus is characterized by the fact that its direction changes when the position of the head is reversed, and it is proposed to exclude it from the present communication. But Nylén also defined another type of positional nystagmus as the "direction fixed" type. In this, certain positions of the head produce nystagmus, and its direction does not change with changes in the position of the head.

Now this, in some respects, corresponds more closely both with what Bárány observed in his case of otolith disease, and with what we have observed in the group of cases now to be described.

It is found, however, on proceeding to this task, that the characteristics of the nystagmus in this, as indeed in any variety of positional nystagmus, cannot be adequately specified in terms of its direction fixation. Furthermore, such a classification takes no note at all of certain other features of the nystagmus which were mentioned very clearly by Bárány and are obviously of great importance; so much so that they must inevitably be taken as our starting point. They are as follows: Firstly,

the character of the nystagmus which is essentially paroxysmal; secondly, the course of the disease which is essentially benign. It is seldom, if ever, we find, associated with any evidence of intracranial disease, and tends to recover with time and simple sedative measures. As will be seen, our evidence goes to show that it is due, as Bárány believed of his case, to a non-progressive lesion of the otolith apparatus. Bárány's own words upon his case may now be quoted. The patient was a 27-year-old woman who had had attacks of vertigo for fourteen days. Hearing was normal, the caloric reactions normal, and the central nervous system normal. Bárány writes:

"My assistant, Dr. Carlefors, first noticed that the attacks only appeared when she lay on her right side. When she did this, there appeared a strong rotatory nystagmus to the right. The attack lasted about 30 seconds and was accompanied by violent vertigo and nausea. If, immediately after the cessation of the symptoms, the head was again turned to the right, no attack occurred, and in order to evoke a new attack in this way, the patient had to lie for some time on her back or on the left side."

Bárány goes on to say that similar observations have been made by himself and others, and the reaction had been attributed to lesions of the semicircular canals. In this case, however, Bárány carried out certain further observations, and demonstrated that the factor precipitating the vertigo was not head movement but head position in space, and for this reason he attributed the condition to a disorder of the otoliths.

Our approach to the study of this condition, of which we have now seen a large number of cases, has been primarily clinical. First comes the matter of symptomatology and in few conditions is careful history-taking of such decisive importance. Apart from the patient's account of his symptoms other points of interest are sex and age incidence, relationship of the symptoms to head injury, to focal infection and to collateral evidence of aural or neurological disease. Finally, note is taken of the duration of the symptoms and their response to treatment.

An otological examination is then carried out with full functional tests of hearing and equilibrium, and lastly an examination is made for positional nystagmus.

Symptoms.—The story given by the patient is characteristically that the giddiness comes on when he lies down in bed or when he turns over in bed, or when such a position is taken up during the day; for instance, in lying down beneath a car or in throwing the head backward to paint a ceiling. The patient sometimes, although not always, recognizes that the onset of the vertigo is associated with this critical position and will say that he does his best to avoid it. He may sometimes also say that he has noticed the phenomenon of adaptation which Bárány described so well in his patients, and can cause his vertigo to disappear by maintaining his head in the disagreeable position, or by taking up this position slowly. The vertigo is essentially transient and it is generally accompanied, but not always, by nausea and, it may be, by vomiting. Cochlear symptoms are generally absent; one other symptom of interest is of discomfort, and it may be of tenderness in the occipital region. Examination of the ears, nose and throat reveals in many cases normal findings, and the same can be said of the usual tests of cochlear and vestibular function. These will be considered after first describing the positional nystagmus and our technique for eliciting it.

The reaction is induced, as Bárány said, by a critical position of the head in space. This can be defined as follows: The patient is laid supine upon a couch with his head just over its end. The head is then lowered some 30 degrees below the level of the couch and turned some 30 degrees to 45 degrees to one side. In taking up this position, the patient is first seated upon the couch with the head turned to one side and the gaze fixed upon the examiner's forehead. The examiner then grasps the patient's head firmly between his hands and briskly pushes the patient back into the critical position. The reaction which results calls for some detailed description.

First of all there is nearly always a marked latent period. Sometimes this is as long as 5/6 seconds. Occasionally it is very short and indeed the reaction may seem to come on at once. This, however, is uncommon. The onset of the nystagmus is nearly always preceded by an appearance of distress. The colour may change; the patients may close their eyes, cry out in alarm and make active efforts to sit up again. At this point it is necessary to reassure the patient and maintain the position of the head. The nystagmus is chiefly rotatory, the direction of the rotation being towards the undermost ear. (*Note.*—In specifying the direction of the rotation reference is made to the displacement of the 12 o'clock point of the corneal circumference.) In addition to the rotatory element there is generally a horizontal component which is again directed towards the undermost ear. The nystagmus increases in a rapid crescendo in a period which may be as short as 2/3 seconds, or as long as 10 seconds. Thereafter it rapidly declines and the patient's distress is relieved. If the patient is then allowed to sit up, a recurrence of the vertigo in a slighter form is generally noted, and if the eyes are examined at this point nystagmus can be seen, the direction of which is, on the whole, reversed. If this is allowed to disappear and the critical supine position is again assumed, the nystagmus again makes its appearance but generally in slighter form and disappears more rapidly than before. After two or three repetitions of this test it is generally found that the reaction has been eliminated altogether and cannot be elicited except, as Bárány pointed out, after a period of rest.

A more detailed consideration will now be given to the oto-rhino-laryngological findings, and the

tests of cochlear and vestibular function. A substantial body of our patients, more than a third, have been found to have entirely healthy ears, noses and throats. Tests of cochlear function have been normal; tests of vestibular function, Romberg, gait and caloric responses have also been normal. Rather more than half of the subjects have exhibited substantial evidence of ear disease. This evidence has generally taken the form of tympanic changes indicative of old and sometimes active catarrhal or suppurative otitis media. In a few cases a severe high-tone deafness has been present alone; in some, due to trauma, in others of obscure ætiology. In most of these, substantial abnormalities of cochlear function, or of the caloric test results were present. In a good many of these cases with ear disease, one ear alone was affected and, as will later be seen, it has been possible to apply this finding to a solution of the problem of localization of the lesion. On the whole, however, it is true to say that evidence of ear disease, although present in some cases, is inconspicuous in the majority and entirely absent in over a third.

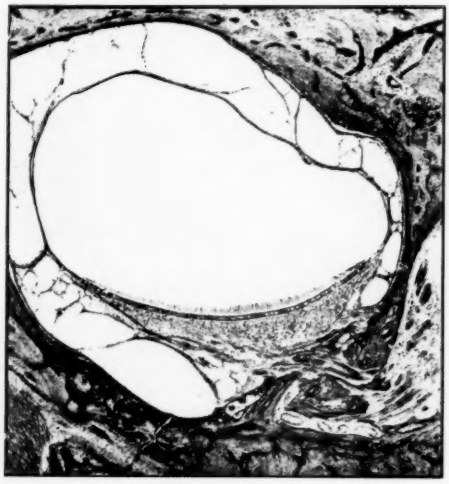


FIG. 8.—(× 15.)

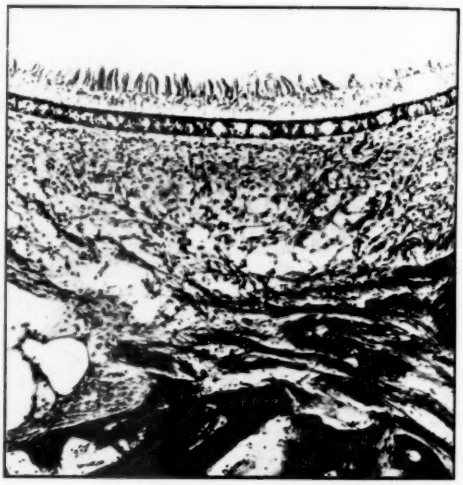


FIG. 9.—(×94.)

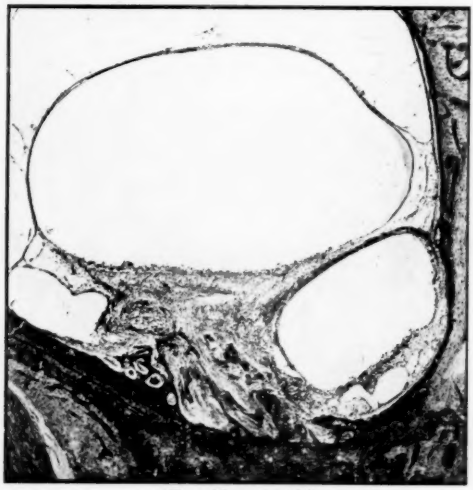


FIG. 10.—(× 15.)

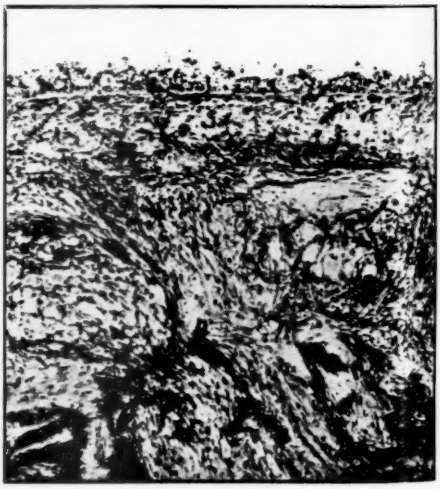


FIG. 11.—(× 94.)

For description of Figs. 8—11 *see* p. 353.

We come, finally, to the course of the disease and the association of the nystagmus with other evidence of neurological disorder. In a good many subjects pain in the neck and occipital region is complained of, and radiological examination has generally revealed some evidence of cervical arthritis. The region, however, affected is chiefly that of the fifth and sixth cervical vertebræ, a very common site in subjects of our predominant age groups, and it has therefore been impossible to attribute any significance thereto. All of our cases have been investigated by our neurological colleagues and, with one or two exceptions of doubtful significance, no evidence has been found of any neurological lesion. The course of the disease is essentially benign. Many of our cases have been followed up for five years or more and in nearly all the symptoms have subsided with sedatives. In a few, infective lesions have been present of the antra or teeth, and these have been eliminated by appropriate surgical measures. It appears likely that the incidence of such infective lesions in our cases has been abnormally high: certainly more so than in cases of Ménière's disease. The evidence for this is, however, inconclusive and it is certainly true to say that in many cases no evidence of infection has been found.

In reviewing the clinical data so far presented it can be said of this disorder that a great deal is now known of its symptomatology and natural history. Much is also known of its physical signs. It is of interest, therefore, to review this knowledge and consider the information which it yields upon the nature of its pathology.

Two things are quite clear. Firstly, the pathological process, wherever or whatever it is, is essentially a benign or self-limiting one. Secondly, the lesion, whatever its nature, is entirely limited to the vestibular apparatus, and here the term "vestibular" is used in its widest sense, to include the labyrinth, vestibular nerve and its central connexions. The questions, therefore, that remain are these: Where exactly is the lesion? and what is its nature?

These we have endeavoured to answer by means of further clinical studies, and by the histological examination of the labyrinth in a human subject.

The clinical evidence will be considered first. The benign character of the disorder and the fact that its manifestations are entirely vestibular exclude at once the possibility that it is due to any destructive lesion, vascular, neoplastic, inflammatory or degenerative within the brain-stem or VIII nerve. If it were, then the occasional occurrence of involvement of other nervous pathways within the brain-stem which lie close to the vestibular neurones would be inevitable. Furthermore, some evidence of the destruction of the vestibular neurones themselves would be apparent, as, for instance, changes in the caloric test results which are so characteristic of vestibular neuronitis and which we have attributed to a toxic destruction of the vestibular neurones within the brain-stem.

It is conceivable, of course, that the nystagmus might be due to some temporary vascular disorder of the brain-stem dependent upon some vascular abnormality. Here it is well known that De Kleyn [18] suggested this very possibility and brought forward good anatomical evidence that in some cases at least an abnormality of one vertebral artery rendered it susceptible to occlusion by certain head positions of the very kind that we employ for eliciting the typical reaction of positional nystagmus. We have been in the habit, from time to time, of examining typical cases without any neck-twisting, and recently we have adopted even more elaborate precautions to exclude this possible source of artefact, if such it could be called.

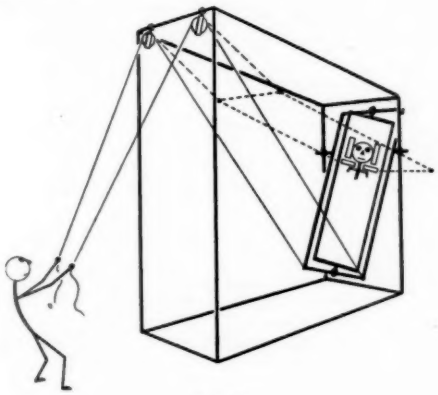


FIG. 7.

By means of apparatus shown in Fig. 7 it is possible to move the patient *en masse* into the critical position, and under these conditions the characteristic nystagmic reaction has been found to make its appearance as usual. It still remains conceivable that some kind of reversible lesion of the central vestibular neurones due

to infection or injury, with or without some anatomical aberration, might bring about the typical reaction to posture now described. Nevertheless, the very fact that the condition *never* exhibits evidence of any extra-vestibular involvement, together with the absence of any evidence of destruction of vestibular neurones, as for instance changes in the caloric responses, makes the possibility of a central lesion exceedingly remote, and on this purely clinical evidence alone we have been inclined to localize the lesion more peripherally, and further, to believe that it has an essentially irritative basis.

Two questions of great interest now arise: Is the lesion in the labyrinth at all? If so, which is the affected labyrinth? And here it has been possible to advance our ideas very considerably by the further analysis of the clinical evidence.

The total number of our cases is 100. Age and sex incidence are given in Table V. The cases have also been divided into three groups in a manner designed to establish the role, nature and localization of ear disease as a possible cause of the positional nystagmus.

The analysis is presented in Table VI.

TABLE V.—POSITIONAL NYSTAGMUS OF THE BENIGN PAROXYSMAL TYPE. 100 CASES

Age Distribution

Age (years)	20–29	30–39	40–49	50–59	60–69	Over 70	Total
Number ..	4	18	27	33	13	5	100

Sex Distribution

Male 52
Female 48 Total 100

TABLE VI.—POSITIONAL NYSTAGMUS OF THE BENIGN PAROXYSMAL TYPE

The Role of Ear Disease as a Causative Factor

	Number of cases
Group I:	
No evidence of ear disease	34
Group II:	
Slight evidence of ear disease (abnormality of caloric responses only) ..	11
Group III:	
Substantial evidence of ear disease (gross middle ear infections, labyrinthine trauma, &c.)	55
Total	100

In the first of these three groups there are 34 cases with no evidence whatsoever of disease of either ear; this group, therefore, provides no evidence of localization. In the next group of 11 cases slight evidence of ear disease was present, and in the majority of these the disease was unilateral. In the third group of 55 cases the evidence of ear disease was more obvious and again, in a large proportion of cases, the disease was unilateral. Correlation of this evidence of ear disease in these two last groups of cases with the direction of the nystagmus, leads to two important conclusions:

(1) That ear disease does play an important role in the causation of the nystagmus.
(2) That the side of the ear disease is related to the direction of the nystagmus in a systematic manner.

Going back to

Group I: In 31 of these 34 cases the positional nystagmus was unilateral, directed towards the undermost ear. In the other 3 cases, the positional nystagmus was bilateral and again directed towards the undermost ear. It is proper to say of these 34 cases that no evidence is provided as to the localization of the lesion responsible for the nystagmus. Further, that the lesion, if it is in one or other labyrinth, has caused no other demonstrable disturbance of its function.

Group II: In all 11 cases the only evidence of ear disease was an abnormality of the caloric test responses. In 4 of these the caloric abnormality was bilateral. In all 4 the positional nystagmus was unilateral and directed towards the undermost ear. In all of the other 7 cases, in which the abnormality was unilateral, the positional nystagmus was also unilateral and directed towards the side of the caloric abnormality when this was undermost. It is necessary to comment on the fact that in all of the 7 of these 11 cases in which an aural lesion was present, even in the form of a caloric abnormality, the positional nystagmus was always directed towards the affected side when this was undermost. This strongly suggests that the lesion responsible for the positional nystagmus was located within the undermost ear.

We come finally to

Group III: The cases in Group III consist of 55 subjects with substantial evidence of ear disease. This evidence is analysed in Table VII.

TABLE VII.—POSITIONAL NYSTAGMUS OF THE BENIGN PAROXYSMAL TYPE

Group III: 55 cases with substantial evidence of ear disease	Bilateral	Unilateral	Direction of positional nystagmus: Towards affected ear when undermost	Towards affected ear when uppermost
A. Evidence of otitis media (suppurative or severe catarrhal)	11	15	15	0
B. Evidence of neuro-labyrinthitis (mumps or syphilis)	1	2	0	2
C. Evidence of inner ear trauma	12	6	5	1
D. Other evidence of ear disease (nerve deafness of obscure ætiology)	7	1	1	0
	31	24	21	3

The 31 cases in which the disease was bilateral can be put aside as presenting no evidence of localization. 24 cases are left in which the ear disease was unilateral. It will be seen that in no less than 21 of these the direction of positional nystagmus was towards the diseased ear when this was undermost. This evidence demonstrates again that ear disease is an important factor in causing the nystagmus. Furthermore, that the nystagmus is directed towards the side of the lesion when this is undermost.

We are thus directed to the conclusion that the lesion *is* a peripheral one and in the labyrinth towards which, when undermost, the nystagmus is directed. Consideration may next be given to the nature of the lesion, and here again clinical studies have proved of decisive importance. The benign course of the disorder makes a neoplastic lesion impossible. The usual absence of cochlear dysfunction and the entirely different character of the vertigo differentiates it completely from the typical hydrops of Ménière's disease, and the remaining possibility is thus of a chronic lesion due to infection, trauma or vascular disease. The lesion certainly affects the otoliths and, since it is so often associated with normal caloric responses, it follows that it is more likely to be irritative in character than destructive.

This concludes our clinical evidence bearing upon the nature and localization of the lesion responsible for positional nystagmus of the benign paroxysmal type.

We have been able to derive supporting evidence from the histological examination of the labyrinths of a characteristic case and an account of our findings is appended.

The case was that of a woman aged 40, a patient of Sir Charles Symonds', who died at the National Hospital, Queen Square, on December 3, 1947. For twenty years she had suffered from vertigo with deafness of unknown cause of the right ear. Her terminal illness and death were due to a glioma affecting the basal ganglia and the upper part of the brain-stem on the left-hand side. On examination a few months before death there was a severe deafness of the right ear of the inner ear type without evidence of tympanic disease. The caloric responses were brisk and normal on both sides and a positional nystagmus of the benign paroxysmal type was present to the right with the right ear undermost. Her symptoms under these conditions reproduced the vertigo from which she had suffered for twenty years. On histological examination the left labyrinth was normal. The right labyrinth showed a severe degeneration of the spiral ganglion in the cochlea which was the essential cause of the deafness. The ampullæ of the semicircular canals appeared normal. In the maculæ of the utricle and saccule, however, very unusual changes were present, in particular the utricle.

In Fig. 8 is shown, for comparison, the appearance of the normal, healthy human utricular macula. The layer of sensory cells is seen evenly arranged with the superimposed otolith membrane. Beneath the layer of sensory cells lies a loose connective tissue meshwork, in which run the fibres of the utricular nerve.

In Fig. 9 is shown a view at higher magnification of the sensory epithelium and the underlying connective tissue meshwork.

In Fig. 10 is shown a view of the macula of the utricle in our case of positional nystagmus. The outstanding feature is the absence of the otolith membrane, the disorganization of the sensory epithelium and certain gross tissue changes in the connective tissue meshwork underlying the epithelium. Its depth is greatly increased. A certain amount of new bone formation has taken place, and at certain points a considerable cellular infiltration is present. This is better seen at higher magnification in Fig. 11.

In Fig. 11 it can be seen that in addition to the absence of the otolith membrane and the disorganization of the sensory cells, there is also present a considerable thickening of the sub-epithelial connective tissue network with the presence here and there of irregular cellular infiltrations. At one point there occur a number of irregular spaces occupied by fluid or cell remnants. The general picture is one of chronic tissue changes resulting either from infection or trauma and it accords very well with our conception of the responsible lesion which we have reconstructed from our clinical evidence. Changes very similar in character but lesser in degree were present in the macula of the saccule.

This concludes our clinical and pathological evidence on the subject of this variety of positional nystagmus. As already stated, it would seem essential to describe it as the benign paroxysmal type

on account of certain outstanding clinical features which distinguish it just as much as its directional characteristics.

In order to achieve clarity emphasis has been laid in the course of this communication upon such facts—on the whole many and weighty—which fortify the central theme of our argument. Certain discrepancies exist; they have not been overlooked and will be given attention in the course of further studies. We do not think that they will be found to invalidate our main conclusion which is that positional nystagmus of the benign paroxysmal type, first described by Bárány in 1921, is due, as Bárány believed, to otolith disease. The lesion consists of chronic tissue changes which may be due to trauma, chronic infection or possibly to vascular disease. It affects, and may be confined to, the sensory epithelium and the sub-epithelial connective tissue of the utricle and saccule of the labyrinth towards which, when undermost, the positional nystagmus is directed.

ACKNOWLEDGMENTS

It is a pleasure to acknowledge the great help that we have received in the course of these investigations from our colleagues, the physicians and surgeons of the National Hospital, Queen Square, who have so kindly put their cases at our disposal; in particular, to Sir Charles Symonds, 2 of whose cases have proved of special importance; also to Dr. C. H. Edwards, who gave us considerable help in the early stages of the preparation and analysis of the clinical findings of our cases of positional nystagmus. Finally, for the construction of the apparatus shown in Fig. 7 and of other special test equipment, and for the preparation and photography of the histological material we are greatly indebted to the laboratory staff of the Otological Research Unit of the Medical Research Council.

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Sir Charles Symonds' paper on "The Significance of Vertigo in Neurological Diagnosis" will appear in the *Journal of Laryngology and Otology*.

[*February* 1, 1952]

The papers by Mr. F. C. W. Capps and Mr. Munro Black on "Glomus Jugulare Tumours" will be published in the *Journal of Laryngology and Otology*.

A paper on "Eosinophil granuloma" read by Mr. A. R. Dingley will also be published in that Journal.

Section of Laryngology

President—F. C. W. CAPPS, F.R.C.S.

[*March* 7, 1952]

DISCUSSION ON STRIDOR IN INFANTS [*Abridged*]

Mr. T. G. Wilson (Dublin): During infancy stridor may be caused by almost any lesion of the respiratory system. The present communication is concerned almost entirely with stridor of intrinsic laryngeal origin, a classification of the causes of which is given in Table I.

TABLE I.—INTRINSIC LARYNGEAL CAUSES OF STRIDOR IN INFANCY

Congenital anatomical abnormalities	Tumours and cysts	Inflammatory conditions	Neurological abnormalities	Trauma	Foreign body
Congenital laryngeal stridor	Papillomata of the larynx	Acute laryngitis	Tetany (laryngismus stridulus)	Birth injury	Vegetable foreign body
Webs of the larynx	Cysts of the larynx	Laryngo-tracheo-bronchitis	Neo-natal tetany		Non-vegetable foreign body
Congenital stenosis of the larynx		Diphtheria Post-diphtheritic stenosis The exanthemata (measles, whooping-cough)	Recurrent paralysis	Injury caused by intubation	

Stridor is the presenting symptom of laryngeal disease in infants, as hoarseness is in adults. This is because of the anatomical and physiological differences between the infantile and the adult larynx. The infant's larynx is relatively smaller in comparison with the body as a whole; the tissues are softer and more flabby; the axis of air-entry is straighter; and the lumen is funnel-shaped, the narrowest point being in the subglottic region about 10 mm. below the vocal folds. In addition to these anatomical differences is the physiological fact that the infant's larynx is easily thrown into spasm by reason of the instability of the infantile nervous system.

The laxity of the laryngeal tissues, when present to an abnormal degree, allows the structures surrounding the *introitus laryngis* to collapse under the pressure of the inspiratory stream, causing congenital laryngeal stridor. When the normal infantile larynx becomes inflamed, as in diphtheria and acute laryngo-tracheo-bronchitis, the already relatively small laryngeal lumen is further greatly reduced by the gross œdema permitted by the loose areolar tissue of the larynx, particularly in the subglottic area. Here again stridor results. A third important cause is spasm, causing laryngismus stridulus.

Congenital laryngeal stridor is said by high authority to be a rare disease. Of 11,076 live births in the Rotunda Hospital during the years 1947, 1948, and 1949, only two cases were diagnosed. It is also said to be rarely fatal. This is true in so far that it is *rarely* a *direct* cause of death, but of 10 patients recently sent to the National Children's Hospital, Dublin, one child died. It is possible that some of the great number of infants whose deaths are reported from "suffocation" or "overlaying" really die from congenital laryngeal stridor.

StClair Thomson and Negus (1948) described the laryngoscopic appearances of this condition as follows: "By means of direct inspection D. R. Paterson and A. Brown Kelly demonstrated that the larynx, as seen clinically, is of an exaggerated infantile type. The epiglottis is very long and tapering and its lateral margins are rolled backwards so as to meet, and thus form a complete cylinder above." This is, of course, perfectly true in a certain number of cases. When, however, Lack is quoted as saying that he has "frequently examined the larynx in children and has never observed

the malformation described except in association with this affection" it must be pointed out that this type of epiglottis is frequently seen in children who have never suffered from stridor. Further, congenital laryngeal stridor is often seen in patients with a normally shaped epiglottis. The theory that congenital laryngeal stridor is caused by an anatomically abnormally shaped larynx, must therefore be rejected for two reasons: first, that many cases showing an "exaggerated infantile" larynx are seen in infants who do not and never have suffered from stridor, and secondly, that a considerable number of cases of congenital laryngeal stridor occur in children with the normal "omega-shaped" epiglottis.

It is obvious that the stridor in these cases results from a valvular closure caused by the concentrated pressure of the inspiratory stream, and that this collapse results from undue flabbiness of the epiglottis and arytenoids. But congenital laryngeal stridor is not of particularly common occurrence amongst premature infants, which suggests that immaturity is not the whole explanation.

Schwartz (1944) considers that the disease is linked with micrognathia, glossoptosis, and other manifestations of arrested development of the mandibular arch. But well-developed micrognathia is not often seen in association with congenital laryngeal stridor, and it would seem that the underlying cause is much more likely to be tracheal stenosis combined with immaturity and softness of the tissues.

The cause of the stridor in congenital laryngeal stridor is therefore passive in character, being produced by the approximation of the flabby laryngeal tissues by the suction of the inspiratory air stream as it passes through the isthmus of the respiratory tract. In *laryngismus stridulus* the reverse is the case, for in this condition the stridor is caused by the spasmodic contraction of the adductor muscles of the larynx. It is merely one of the signs of a much larger clinical entity: tetany, or spasmophilia, resulting from rickets.

Rickets is not a common disease nowadays, but between the years 1942 and 1948 there was in Dublin a serious increase in its incidence, brought about by the use of the whole wheat-grain in making bread. The husk of the wheat grain contains phytic acid, which combines with ingested calcium to form an insoluble compound, which is excreted. As a result there was in Dublin a definite increase in the incidence of rickets amongst the children of the so-called "working-classes". Amongst the cases described were a number who developed spasmophilia.

The attack of laryngismus stridulus occurs suddenly, often at night. The child, who was previously lying quietly in bed, is suddenly afflicted with great respiratory distress. He sits up in bed clutching at the sides of his cot for support, struggling and gasping for breath. All the accessory muscles of respiration are called into play—the chest heaves, the nostrils widen, and the pupils dilate. The face becomes blue, and air is sucked in with loud crowing respirations. After a time the child takes a long deep breath, and recovers. Chvostek's sign, Trousseau's sign, carpo-pedal spasm, opisthotonos, incontinence and other unpleasant manifestations may also occur.

As the incidence of rickets in older children diminishes, that of tetany with stridor in the newborn seems to increase. In the Rotunda Hospital, during the years 1947, 1948, and 1949, of 11,076 live births, 48 developed neo-natal tetany. Of these 48 cases, only one developed laryngismus stridulus, which seems to be a contrast to the experience elsewhere.

The symptoms of tetany neonatorum are quite well defined. As they yield rapidly to appropriate medical treatment with calcium gluconate, vitamin D, sedatives, and parathyroid extract, the condition is of little interest to laryngologists except in the matter of diagnosis.

In laryngismus stridulus, therefore, the stridor is mainly caused by spasm. In the stridor of *acute laryngitis* of childhood another factor comes into play, for it is caused partly by spasm and partly by inflammation. These children are usually older, between the ages of 3 and 8, and with tactful persuasion it is usually possible to get a good view of the larynx with the laryngeal mirror. The larynx as a whole will be seen to be injected and hyperæmic, but the striking feature is the "beefy-red" subglottic swelling.

Acute laryngo-tracheo-bronchitis is a disease which mainly affects children under 3 years of age. It is characterized by progressive laryngeal obstruction, and differs from diphtheria in that the mucosa is more œdematous and the secretion thick and tenacious. There is a tendency to the formation of inflammatory crusts in the air-passages, which often results in bronchial plugging and atelectasis. It is a serious disease, difficult to treat, with a high mortality.

The onset of this condition is insidious, and unlike laryngeal diphtheria, which, with vegetable tracheal foreign body, is the principal item of differential diagnosis, it is not rapidly progressive. Dyspnœa with recession of the lower intercostals is the cardinal feature. Croupiness or a hoarse cry is not an early symptom, nor is cyanosis. The stridor is characteristically inspiratory and is sometimes crowing. The primary feature in the production of stridor in these cases, as in acute laryngitis, is the relatively enormous swelling which takes place in the region of the *conus elasticus*. As Negus has said, a swelling of 1 mm. in the mucous membrane is a very serious matter in a child whose subglottic lumen is normally 6 or 7 mm. in diameter.

In acute laryngo-tracheo-bronchitis the subglottic mucous membrane is not only swollen but is also ulcerated and covered with a sloughy grey membrane. The trachea and bronchi are

predominantly involved, and pneumonia and atelectasis from plug formation form a prominent part of the clinical picture. Progress normally is slow, but occasionally one meets a fulminating case in which the infant is rapidly prostrated by an overwhelming infection.

The medical treatment of acute laryngo-tracheo-bronchitis with chemotherapy and humidification raises points of great interest, but the instrumental treatment by tracheostomy and bronchoscopy is more the laryngologist's concern. If the obstruction is severe tracheostomy should be done before signs of exhaustion appear. It must be remembered that the obstruction to breathing in these cases may be as great below the tracheostomy as above it and that the primary object of the operation is in most cases to facilitate bronchoscopic suction and lavage.

The symptoms of acute laryngo-tracheo-bronchitis may be summarized as those of acute infection *plus* endogenous foreign-body formation. It is, therefore, easy to see how the presence of an exogenous vegetable foreign body in the air-passages may cause an error in diagnosis, particularly if inflammation should supervene. Vegetable foreign body in the trachea or bronchi is, indeed, often given as an item in the ætiology of the disease, although its proper place is in the differential diagnosis. A vegetable foreign body which breaks down and becomes infected may, indeed, cause inflammation of the air-passages, with plugging and atelectasis, but acute laryngo-tracheo-bronchitis is a specific infection caused by an organism, probably a virus, which has not yet been identified.

In conditions such as *congenital web* and *papillomatosis* the stridor may be either inspiratory or double. This variation in the timing of the stridor is interesting, for it may be a guide to the site of the obstruction. Generally speaking, an obstruction to respiration at or above the vocal folds will cause inspiratory stridor, while below that level it is usually double or expiratory in timing.

In congenital laryngeal stridor, whooping-cough, and laryngismus stridulus, the stridor normally is inspiratory. In acute laryngo-tracheo-bronchitis also the stridor usually is inspiratory, although the obstruction here is partly, indeed mainly, subglottic. Thymic obstruction will also at times cause an inspiratory stridor, which differs from that of congenital laryngeal stridor in the important fact that it is not present during sleep (Norris, 1949). Supraglottic causes of obstruction will also cause inspiratory stridor; as, for instance, lingual cysts and choanal atresia, in the latter of which a peculiar clicking noise on inspiration is described. The newly born infant's urge to breathe through the nose is tremendous; when it is impossible to do so he still makes violent efforts at nasal respiration. He turns blue and chokes, making ineffectual efforts to breathe; finally he manages to take a breath through his mouth, and in the transition from attempted nasal to oral breathing this "click" is made. A similar mechanism may be observed in dermoid polypus or "hairy dermoid" of the nasopharynx which is also a disease of the newborn.

When the obstruction to respiration is in the trachea, the picture changes. The stridor is now either double or predominantly expiratory, as in tracheomalacia, vascular ring, tracheal and bronchial foreign body. A pronounced expiratory stridor may be heard in cases of pneumonia in which there is no laryngeal or tracheal inflammation. Chevalier Jackson's (1936) "asthmatoid wheeze" in cases of vegetable foreign body in the air-passages is another example of expiratory stridor caused by a low obstruction.

Table II is a summary of the characteristics of the stridor in various intralaryngeal conditions.

TABLE II.—CHARACTERISTICS OF STRIDOR IN VARIOUS INTRALARYNGEAL CONDITIONS

	Direction	Sustained or spasmodic	Age of onset	Presence during sleep	Voice
Congenital laryngeal stridor	Inspiratory	Sustained	From birth or soon after	Diminished	Clear
Laryngismus stridulus	Inspiratory	Spasmodic	From six months to two years	An attack will waken the patient	Clear
Tetany neonatorum	Inspiratory	Spasmodic	Soon after birth	Attack awakens patient	Clear
Pertussis	Inspiratory	Spasmodic	Variable	Attack awakens patient	Clear
Acute laryngitis	Inspiratory	Spasmodic	From three to eight years	Occurs most often at night	Hoarse, strong
Acute laryngo-tracheo-bronchitis	Inspiratory or "two-way"	Sustained	Variable	Yes	Hoarse
Diphtheria	Inspiratory	Sustained	Variable	Yes	Hoarse,
Congenital web	Inspiratory or "two-way"	Sustained	Soon after birth	Yes	Weak
Papillomatosis	Inspiratory or "two-way"	Sustained	Not usually before 2 years of age	In severe cases	Hoarse

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Dr. A. White Franklin (London): The pædiatrician takes a light-hearted view of the baby presenting with laryngeal stridor unless there is obvious failure to thrive or unless there are evidences of respiratory obstruction. Since many mild cases begin to be noisy from birth, or the first few days of life, there is a theoretical differential diagnosis of neonatal conditions associated with stridor on breathing, such as asphyxia, atelectasis, intracranial hæmorrhage, pneumothorax and diaphragmatic hernia. In these, as in micrognathia, the clinical picture makes it certain that the condition is not primarily one of congenital laryngeal stridor. A retention cyst in a mucous gland in the epiglottic wall may cause severe breathing and feeding difficulty. If the possibility be remembered, diagnosis and treatment are easy, though a tracheotomy may be necessary. Diphtheria is an extremely rare possibility, neonatal tetany is a somewhat doubtful diagnosis and congenital heart disease or congenital vascular anomaly may be the cause of the stridor.

Observations on the upper aperture of the larynx have been made by laryngoscopy since the first description by Lees (1883), and Schwartz (1944) has described a motion picture showing indrawing of the upper aperture with inspiration. Thomson and Turner (1900) reproduced collapse of the upper laryngeal aperture by a study of fifty larynges from post-mortem of infants and children and blamed the softness of the parts and inco-ordination of respiratory movement, while Sutherland and Lack (1897) regarded the condition as due to a congenital malformation. Gabriel Tucker (1932) gave measurements of three diameters of the glottis making 5 mm. the minimum compatible with life and development in a normal-sized infant. Iglauer (1922) stopped stridor by guillotining the epiglottis. Finlay (1949) described 3 cases in a family of 4 with a different local cause in each. Many papers have reported abnormal X-ray appearances of the upper mediastinum in relation to congenital heart disease and the condition of the thymus.

Supraglottic obstruction is nearly always due to the peculiar behaviour of the upper laryngeal aperture. Micrognathia produces obstruction because the tip of the tongue somersaults into the pharynx, or its base presses the epiglottis down and back. A cyst arising from the thyroglossal duct and a retention cyst in the epiglottic wall may produce the same effect. Lesions of the vocal cords that have been described are, papilloma, cysts and congenital webbing, double abductor paralysis and laryngismus stridulus. Subglottic obstruction may be due to mucosal hypertrophy (Hamme, 1946), or the presence of some tumour including a hæmangioma. Chronic laryngitis, tuberculous laryngitis, laryngitis stridulosa are also causes and "laryngeal stridor" may result from pressure on the trachea by a mediastinal tumour, enlarged tracheo-bronchial glands, enlargement of the thyroid or the thymus. Softness and collapsibility of the trachea have also been observed. Congenital anomalies of the great vessels pressing on, or forming a ring round the œsophagus and trachea, produce stridor and feeding difficulty in some babies, and congenital heart disease occurs in a definite proportion of cases clinically identical with congenital laryngeal stridor. Mercer (1945) has recorded acute stridor and respiratory failure following successful surgical treatment for œsophageal atresia.

The thymus has been blamed by many observers for laryngeal stridor. A survey of the literature and of cases suggests that though theoretically possible this is very rare and further that it is dangerous to accept the thymus as a cause where there is serious obstruction without the very fullest examination of the larynx. Much of the difficulty has been caused by failure to recognize the appearances of the normal thymus in chest X-rays (Pancoast, 1930; Kemp *et al.*, 1948). The upper mediastinal shadow alters so greatly between inspiration and expiration that unless at least eight intercostal spaces are seen clearly above the diaphragm, little notice should be paid to apparent increases.

The records of 30 cases that fall into the clinical group of congenital laryngeal stridor have been analysed. These attended St. Bartholomew's and Osterhills Hospital. All but 5 of these I have seen myself. 16 were males, and 14 females. Only 3 were severe, 2 of whom had laryngeal abnormalities, and 27 were mild from the point of view of the stridor. 10 were hard to rear owing to breathing or feeding difficulties or both, or because of serious acute illness. In the remaining 20 there was never at any time cause for anxiety. Stridor ceased by the age of 2 years in 10 cases, by the age of 3 in 7, 6 are still stridulous but these are under 3 years of age. 4 cases continued through childhood, 2 ceasing in late childhood, and 2 are now aged 4 and 7 years. 1 infant has lost contact. 3 died in infancy, each with a normal larynx at post-mortem examination, 1 of pneumonia, 1 of gastro-enteritis and 1 of a bleeding duodenal ulcer. Onset was at birth in 15 and within the first month in 6 cases, being later in 9. All had inspiratory stridor, 6 had expiratory stridor as well and 14 had in infancy signs of obstruction to respiratory movement, suprasternal recession and indrawing of the lower chest with inspiration. Alæ nasi did not work. Cyanosis occurred in occasional attacks in 5 cases, usually at a time of respiratory infection.

The chest picture was normal in 16 out of the 23 cases X-rayed. In 7 there was an unusual shadow either of an obvious thymus (4) or of cardiac enlargement (3). The barium swallow was normal in all cases so examined. Laryngoscopy was undertaken in only 11 cases, the appearances being normal in 6 and abnormal in 5. The small proportion of laryngoscopies reflects the mildness of the condition in the majority of cases rather than unwillingness to consult the laryngologist. In 2 the upper laryngeal aperture was described as floppy, in 2 there was subglottic swelling of undetermined nature and in one there was general narrowing of the larynx and upper trachea thought to be due to mucosal hypertrophy. 10 cases had repeated respiratory infection. 2 children only have small lower jaws. 3 of the children were adopted. There is a family history in 2 cases, of 3 stridulous infants in one and 2 in another, only 1 from each family being included in the series. Family history is negative in 15, unknown in 4, and the affected is an only child in 9 cases.

3 special cases of laryngeal stridor not included in the series were: 1, a 10-month-old baby presenting with inspiratory stridor and later dying of miliary tuberculosis with tuberculous laryngitis and large tracheo-bronchial lymph nodes. The second, a case of micrognathia with a cleft soft palate, extremely difficult to manage at first but now at 11 months weighing 18¼ lb., the cleft palate having been recently repaired by Mr. Elliot-Blake; and the third, a case of Dr. Everley Jones' (Dolton and Jones, 1952) of a baby with a double aortic arch, the stridor having been relieved by dividing the narrow left aortic arch and the ligamentum arteriosum (Gross and Neuhauser, 1951).

Three groups of cases may be distinguished, firstly, cases where the lesion is found to be some anatomical abnormality, often a kind that may be free from symptoms. These cases usually arise during the first few months of life and are likely to be associated with inspiratory and expiratory stridor, hoarseness of cough or cry, and cyanotic attacks. Complete examination is essential. The second type is the commonest, where laryngeal stridor is due to a floppy, easily obstructed upper laryngeal aperture. The baby is completely healthy, and, apart from the noise, gives no cause for anxiety. Cough and voice are normal. There is inspiratory recession but no gross respiratory obstruction. The noise increases for four or five months, is made worse by crying, emotion and feeding and continues in sleep, becomes intermittent and disappears altogether during the third year of life. In the third group, the definite feature is not the quality or the character of the noise so much as the general feebleness of the child who is difficult to rear and tends to recurrent respiratory infections. Such babies may die suddenly and rather surprisingly from respiratory or gastro-intestinal infection. Though rare, the existence of this group must be remembered when prognosis is in question.

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Mr. Charles E. Scott (Edinburgh): From records of 40 cases in the Royal Hospital for Sick Children and City Hospital for Infectious Diseases, only 10 cases were diagnosed as congenital stridor. In many of our textbooks only two types of stridor are described: laryngismus stridulus and congenital laryngeal stridor. The former is met with in children under 2 years of age, and is characterized by its sudden onset and spasmodic nature, being generally associated with rickets and malnutrition.

A typical case of congenital stridor is characterized by an inspiratory croaking sound, which is noticed immediately after birth. The croaking is more marked during waking hours and may be aggravated by excitement. There is also indrawing of the thoracic and abdominal walls, evidence of inspiratory obstruction. This difficulty does not appear to alarm the child in any way and there is little tendency to cyanosis. The stridor may become more marked during the first few months of life, then subside; and then following a quieter period, gradually disappear by about the age of 2 years. There is seldom any call for treatment, but one must reassure the parents. The infant in all other respects is perfectly healthy.

The cases discarded were almost all due to some acute infection which could generally be demonstrated; but congenital stridor still requires clarification. Many theories have been advanced. Amongst them is that of pressure on the trachea by an enlarged thymus gland (Avellis).

McBride describes it thus: "An extremely interesting affection, allied to laryngismus stridulus, and yet differing from it in some important characteristics, is the condition known as infantile respiratory spasm. It occurs, either immediately after birth, or within a few days or at the most weeks. The infant more or less continuously croaks and crows, but on close inquiry it will be found that certain periods of intermission occur from time to time. Cases of this nature have been described by Lees, Gee, and Löri, the last-named claiming to have observed with the laryngoscope periodic closure of the vocal cords. Lambert Lack also made laryngoscopic examinations in 6 cases, and states that the epiglottis was always folded upon itself, and the aryepiglottic folds approximated. To John Thomson, however, we are indebted for not only generalizing from the statements of others, but placing on record a series of most carefully observed cases which came under his notice. According to this author, the patients are usually free from rickets, the cry is generally quite clear, there is marked indrawing of the chest and episternal notch on inspiration, and in some of his cases there was evidence of commencing pigeon-breast. There is withal a marked absence of cyanosis and even distress, while the alæ nasi move little, if at all. The stridor goes on during yawning, suckling and while the tongue is depressed. I have also observed such cases, and looked upon them as similar to laryngismus. Probably this is correct, in so far as both are due to cortical irritation; at the same time it must be admitted that the clinical differences are marked" (McBride, P. (1900) Diseases of the Throat, Nose and Ear. 3rd edit. Edinburgh and London).

Here we have a very interesting picture of the condition we know as congenital laryngeal stridor, together with the suggestion that there was some cortical lesion. This idea is further stressed in the theory advanced by Logan Turner and John Thomson. They thought that owing to a late development of the cortical centres controlling respiration, a resultant inco-ordination of respiratory movements developed. Thus there was established a rhythmic sucking in of the soft structures forming the upper aperture, which resulted in an accentuation of the infantile larynx.

The stridor is phonatory in nature and, therefore, is not produced in the pharynx; movements of the tongue or mouth or occlusion of the nose do not affect it. Adenoids were not thought to act as a reflex source of irritation. Lack and Sutherland considered the laryngeal deformity was not primary, but acquired as a result of the inco-ordination of the respiratory movements, that is, that the diaphragmatic intracostal inspiratory movements are not synchronous with those of the laryngeal abductors.

We all know the pattern of the infantile larynx, particularly the long tapering epiglottis with its enfolded edges, the narrow chink resulting from the approximation of the aryepiglottic folds, in all forming almost a complete tube. By normal growth we know how the larynx expands and the epiglottis gradually unfolds and opens out. Have we here the true explanation of the stridor? Have these theories of inco-ordination been proved?

In the past, I must confess to having diagnosed congenital stridor without having carried out a full laryngoscopic examination, when confronted with a croaking but otherwise healthy infant. Every case should be examined with the laryngoscope under a general anæsthetic, which permits the operation to be leisurely and complete. In all cases I have noted the infantile type of epiglottis or an exaggerated form. I concluded that the answer we seek is largely an anatomical one. I think that the exaggerated infantile type of larynx is an accepted fact, and also that the infant is perfectly healthy in other respects.

Have the inco-ordination theories and their part in the condition been proved? If not, some research with modern electrical apparatus might be worth considering.

Still to be answered is why, in a small number of otherwise healthy infants, do we find this soft flabby larynx. What causes the degree of flabbiness required to produce this stridor? Is it due to some metabolic fault such as calcium deficiency?

One would suggest that given the necessary degree of softness in an exaggerated infantile type of larynx, there is an analogy between the epiglottis and aryepiglottic folds and the reed placed in wood-wind instruments; the function of the reed being to set the column of air vibrating; the same effect produces stridor in infants.

Dr. J. F. Birrell (Edinburgh): The erroneous belief that an infant is an adult in miniature leads to difficulties in training oneself for bronchoscopy in babies. There is no adequate description of the

normal endoscopic findings in infancy. There are many instruments devised for endoscopy in the adult, which have no counterpart for the child or baby, yet the need for endoscopy, and often emergency endoscopy, in infancy is becoming more frequent.

This paper is based on all the cases of stridor with which I have had personal contact during the years 1946–51. I have discarded all cases of acute stridor, and am left with 25 cases, the grouping of which is shown in the table.

CHRONIC LARYNGEAL STRIDOR (25 CASES)

Congenital laryngeal stridor	13	Hypertrophy of ventricular bands ..	2
Paresis of vocal cord	2	Congenital laryngeal papillomata	1
Congenital laryngeal web	1	Subglottic causes	6

The diagnosis of congenital laryngeal stridor should never be made without laryngoscopy under anæsthesia. When no anæsthetic is given one may see little beyond a laryngeal spasm and a few convulsive inspirations. I cannot submit to the theory that one can safely bronchoscope a conscious infant without pain, shock and trauma.

Every case is assessed with the anæsthetist, who must guide the endoscopist during the examination as to the infant's safety, as the pulse, the colour and the ventilation are the criteria of danger. Every baby should be anæsthetized unless the indrawing is so marked, or the secretion so profuse, as to suggest that there is greater danger from the loss of its reflexes than from the examination.

For laryngoscopy alone a rapid induction with ether will result in equally rapid recovery, but where bronchoscopy is envisaged, chloroform, with its more prolonged action, is added. This has the extra advantage that it produces an analgesic effect, not seen with ether, which permits inspection of the cords in a subdued infant with active reflexes.

The larynx in a congenital laryngeal stridor is extremely mobile, and is tucked forwards below the tongue. It swings upwards and forwards on inspiration while the aryepiglottic folds come together. The fact that stridor persists under anæsthesia, even when this is taken down almost to respiratory arrest, not only negatives any cortical cause, but enables the operator to determine at what level the stridor is produced, by noting when the noise disappears. In this connexion it is important to remember, particularly if the baby shows some glossoptosis, that the epiglottis cannot be elevated without at the same time elevating the tongue.

Should the stridor persist after elevating the epiglottis, the cause may lie in the larynx itself, or below the glottis. The cases with laryngeal involvement require little comment save that, if there be paralysis of the vocal cord, the presence or absence of sensation must be tested for. If there is anæsthesia, the baby must always be fed, and laid in its cot, with the affected side uppermost, lest any milk or vomit enter the lung over an insensitive cord.

The congenital papillomata had been present since birth, although the infant was five months old when first examined. Aureomycin has produced no effect, and the condition is progressing. It is the only case in the series who has not recovered from the stridor.

There have been 6 cases of chronic subglottic stridor, all in babies with an X-ray report of enlargement of the thymus. Each had a normal larynx with some redundancy of the subglottic tissue. On bronchoscopy the posterior tracheal wall bulged forwards into the lumen of the trachea on expiration, almost obliterating it. The main bronchi showed the same findings, resembling the picture seen in asthma. All recovered within a year. Only 1 infant had deep X-ray therapy to its thymus, and this so increased the subglottic œdema that tracheostomy was required.

It would appear that there is a common pathology, and that this is in some way associated with the thymus. There was no narrowing of the lumen by pressure as used to be taught. It may be that the presence of an enlarged thymus produces an increase in the intrapleural pressure, as this has been shown by Jenkins *et al.* (1950) to give rise to similar bronchoscopic appearances in adults; or the thymus may merely produce an increased cough reflex, which has been shown by Jackson to cause a bulging forwards of the œsophagus into the trachea.

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The President said he could confirm what Dr. Franklin had said about the role of the pædiatrician in cases of laryngeal stridor in infants. Tomograms had given valuable information in the adult larynx and, if practicable in infants, it might help to elucidate some of these conditions.

Mr. V. E. Negus thought it might be desirable to reclassify some of the causes of stridor in childhood, in view of the opinions expressed by Mr. Wilson.

Mr. Wilson had agreed that a limited number of cases were due to congenital narrowing of the laryngeal aperture and these would fall under the heading of congenital laryngeal stridor.

Others were ascribed to spasmodic contraction of the sphincteric group of laryngeal muscles in tetany, a condition observed by Mr. Wilson and apparently well established; this could be described as spasmodic laryngeal stridor.

A third cause was sucking in of the aryepiglottic folds and epiglottis, resulting from undue laxity of the tissues or, in Mr. Negus's opinion, to feebleness of the posticus muscles, which normally should

brace back the arytenoid cartilages and keep taut the aryepiglottic folds, which were supported by the prop-like cartilages of Wrisberg. This condition could retain the name of laryngismus stridulus.

With regard to bronchoscopes for use in very young infants, the speaker's suckling type, in frequent use, seemed to meet all requirements.

Its construction allowed it to have a very small external circumference of 15 mm. and a sufficiently wide lumen, owing to the slender light carrier and the groove in which it was held, instead of a tube.

There seemed no necessity for the construction of different instruments or for their purchase abroad.

Mr. James Crooks said that one of the speakers had insisted that general anæsthesia was necessary in these examinations. He certainly endorsed that view. There should be no bronchoscopy or laryngoscopy in children without it. In his department they saw a good many so-called laryngeal stridors due to the exaggerated infantile type of larynx, and he thought that all children with stridor should be laryngoscoped. During the last year they had discovered two hæmangiomata in the larynx and two cysts in the arytenoid-glottic folds, and the symptoms in these cases were very like those attending "congenital laryngeal stridor". They might easily have been missed.

Mr. W. O. Lodge hoped that those revising laryngological textbooks would keep the taxonomy as simple as possible. He felt that neonatal lives could be saved with the aid of 3 mm. bronchoscopes. At eighteen months, a 5-mm. tube could be passed. Many foreign bodies were now of radiotransparent plastic material.

Dr. B. McNicholl suggested that most cases of acute laryngitis in children were unassociated with marked changes in the trachea or bronchi.

It was important in treatment of inflammatory laryngeal obstruction, particularly where there did not appear to be a tracheo-bronchitis, to avoid the use of oxygen tents. The reasons were twofold. Firstly, the temporary improvement could mislead one and cause unwise delay. Secondly, if signs of anoxia and cyanosis recurred in the oxygen tent, removing the child then for a tracheotomy often caused a fatal collapse before the airway had been restored, due to the transition from a high oxygen concentration to the atmospheric one. The right course of action includes the use of steam, constant and experienced observation and tracheotomy at the optimum time before severe anoxia and circulatory collapse supervened.

He was indebted to Dr. C. J. M. Laurent of the Park Fever Hospital for most of these views.

Miss Josephine Collier said she had recently seen an infant *in extremis* from a laryngeal cyst. The child had had noisy breathing and difficulty in feeding, sucking eagerly but quickly stopping and gasping for breath. Choanal atresia had been considered but a fine catheter passed easily through the nose into the pharynx. When Miss Collier saw the child he was grey from failing respiration. Direct laryngoscopy showed a large cystic swelling apparently arising from the left aryepigiottic fold and occluding the entrance to the larynx. An infant bronchoscope was passed without difficulty but the respiration did not improve. She exhibited the post-mortem specimen which showed how the cyst was responsible both for stridor and for difficulty in feeding and how early direct laryngoscopy would have led to a correct diagnosis and aspiration of the cyst before respiration failed.

Mr. W. A. Mill commented on the relative rarity of cases of retropharyngeal abscess, which were seen at fairly regular intervals a number of years ago. In his experience the condition seemed to have become much more uncommon.

The President said that he did not think he had seen such a case for a very long time He wondered whether its rarity had anything to do with the use of antibiotics.

Mr. E. H. Rainer said that he had seen 2 cases in recent years—one of them two years ago, and the other more recently. Both these cases gave a considerable amount of trouble, but he was glad to say they both lived, though the stridor was very severe. Another case in which stridor was found was one of laryngeal abscess following an acute tonsillitis. The stridor was severe and the patient nearly died.

Mrs. Florence Cavanagh said that no one had mentioned stridor due to impacted foreign bodies in the upper end of the œsophagus. She had seen 3 cases in the last three years. Each was admitted to a medical unit and diagnosed as pneumonia or laryngo-tracheo-bronchitis.

When no improvement occurred after medical treatment X-rays were taken. One revealed a half-penny which, on removal, was markedly corroded. Another was due to a piece of mutton bone. The third case was especially interesting as X-ray showed a small metal foreign body shaped like a question mark and obviously too small to cause such symptoms. On œsophagoscopy a firmly impacted plastic overall-button was removed. The plastic part was grossly swollen, but the only part showing on X-ray was the metallic portion which attaches the button to the coat.

In these cases the symptoms were entirely from the respiratory system and the physicians were utterly surprised by the X-ray.

Mr. Philip Scott (Exeter) mentioned a case in which, on X-ray evidence, the thymus appeared to be enlarged, but it was afterwards discovered that the shadow which the radiologist had attributed to the thymus was caused by a fold of skin at the back of the child's neck during a slight twisting of the body in the examination.

United Services Section

President—Sir GORDON GORDON-TAYLOR, K.B.E., C.B., F.R.C.S.

[*March* 6, 1952]

DISCUSSION ON STANDARDS OF FITNESS IN THE SERVICES FOLLOWING MAJOR SURGICAL PROCEDURES

Air Commodore F. W. P. Dixon, R.A.F.:

In England the assessment of fitness for service by appointed and authoritative bodies has existed and has produced problems of surgical interest for hundreds of years, and no doubt certain agreed physical standards were necessary to qualify a candidate for active service even in those far-off days.

Following the granting of a Charter by George III to the Royal College of Surgeons in London, it soon became the duty of the Court of Examiners "to examine every person who shall be a candidate to be approved to serve as a surgeon or assistant surgeon in any Regiment, Troop, Company, Hospital or Garrison of soldiers, or to serve as surgeon or surgeon's mate appointed on board any ship or ships".

In addition to the above duties, however, this distinguished Court was also required to function in a manner comparable to our modern medical boards, in so far as it investigated compensation and superannuation problems, one of the most famous of which was that of Admiral Lord Nelson's injuries.

With the advance of science both in medicine and warfare and the advent of aerial combat, the physical assessment of a candidate now calls for not only a thorough clinical examination but also an intimate knowledge of that monumental publication the "PULHEEMS".

It has been asked on more than one occasion why major surgery, e.g. gastrectomies, laminectomies, &c., should be performed on serving personnel. Are the individuals fit for service as a result of such procedures? Is there sufficient material to justify such routine operations in Service life? I think the answers to these queries are three in number:

(*a*) Saving of man-power,
(*b*) The provision of surgical experience in the Services, and
(*c*) The social problem involved.

(*a*) *Man-power.*—In the Services, particularly in a specialized or highly technical Service, we have personnel who have required many years of training and apprenticeship before they become proficient at their jobs. We also know, to take a specific and common disability today, that the affliction of *peptic ulceration* is usually confined to the lower middle-age group. These are the very people who have been trained as specialists or technicians in the Service, and who at the peak of their usefulness and ability become invalids or semi-invalids, leading a life of dieting (together with untold quantities of alkalis) or repeated treatments in hospital. We feel that where adequate medical treatment has failed to cure this condition, surgery, and not invaliding from the Service, is the answer to this problem.

(*b*) *Surgical experience in the Services.*—Service surgery in peacetime should not be *confined* to training in war surgery, traumatic surgery or minor operations. A surgeon, to be an efficient and capable wartime surgeon, must of necessity have had a thorough and extensive experience of major general surgery. This can be acquired only by practice and experience in the out-patient clinics, wards, operating theatres, and follow-up systems in a busy general hospital.

(*c*) The social problem to-day is a very real one, namely the increased burden of civilian out-patient clinics, and the long waiting lists for admission to hospital for treatment. Cases invalided from the Service become an added liability to our civilian colleagues, and augment the already crowded waiting lists for admission to civilian hospitals. In fact, at several of our Service hospitals today we admit surgical cases from civilian waiting lists. This, in addition to assisting to clear such lists, also enhances the value of our hospitals as training centres for junior surgeons by providing a wider range of work and experience than might otherwise be their lot.

The most *important* reason for performing these operations is, however, that of saving trained personnel for further service. Tables I to V show clearly that such saving of man-power has been achieved as a result of major surgery.

I shall limit my discussion to two disabilities, their treatment and results. These are:

(*a*) Peptic ulceration, and
(*b*) Prolapsed intervertebral disc.

(*a*) *Peptic ulceration.*—In past years it has been the usual practice to invalid from the Service a man who has repeatedly broken down and failed to respond to courses of medical treatment in hospital, and, in fact, who was a proven case of chronic peptic ulceration and unfit for a full Service life. As I have stated above, these individuals were on the whole confined to that group of trained and valuable personnel, and such a wastage was a serious one. Quite apart from the man-power question, I also feel it is our duty as medical men to offer them a chance of cure and freedom from their disease, whether they be Service personnel or civilians.

Our present practice, therefore, is to *consider* surgery for any individual who has developed symptoms following two separate periods of medical treatment in hospital.

A routine procedure is to perform a partial gastrectomy using an anterior Polya method of reconstruction. It has not been the practice to use a valved stoma in the anastomosis, and our numbers of "dumping" syndromes have been almost negligible. The patient is usually out of hospital in three weeks and, following a period of twenty-eight days' sick leave, he returns to some form of light duty. Three months or thereabouts after the operation he returns to hospital for a review. This consists of a full clinical examination, barium meal and test meal. These investigations being satisfactory, he returns to duty but in a "home service only" category. In the case of aircrew they are still grounded. Aircrew are reviewed again in hospital at the end of six months after operation, and, if satisfactory, are returned to limited flying duties as second pilot and restricted as to height and duration of flight.

All cases, both ground personnel and aircrew, who twelve months after partial gastrectomy for ulcer are found to be symptom free and who give satisfactory results on barium meal and test meal are returned to their full duties. There have been a few selected cases in aircrew who returned to their full flying duties in six or nine months. Table II comprises individuals who have already completed twelve months or longer since their operation. By following our routine and post-operative follow-up of these cases we have found that twelve months is a reasonable period at which to consider upgrading the medical category. All cases are reviewed at the end of a further period of twelve months, i.e. two years after their operation.

(*b*) *Prolapsed intervertebral disc.*—This somewhat fashionable lesion of modern times has now presented itself as a problem in the Services. When one visits rehabilitation centres and conducts out-patient clinics today the thought occurs to one: what happened to all these low back pains and so-called "sciaticas" in young people in days gone by? I feel that the term "P.I.D." has become somewhat abused and too often as an escape mechanism in a difficult case, or when the case is just not diagnosed. However, the classical syndrome of a prolapsed disc occurs fairly frequently and eventually, if conservative treatment fails, is then presented for surgery.

Treatment

(1) *Conservative.*—This is carried out by our orthopædic colleagues and consists of

(*a*) Initial strict bed rest for a period of three to five weeks. *I stress strict bed rest.* If improvement results, then the patient is mobilized either in a plaster jacket or in a Goldthwaite belt.

(*b*) If symptoms recur, then a further period of rest.

(*c*) If no improvement is obtained from (*a*) or (*b*), or if the condition recurs for the third time, then surgery is considered.

(2) *Surgery.*—Our routine in surgical treatment is as follows:

A hemilaminectomy exposing at least two nerve roots on the affected side. Removal of prolapsed or degenerate nucleus pulposus, and freeing the nerve root of adhesions.

No spinal fusion is performed for the following reasons:

(*a*) Results show that of all cases after operation a very small percentage, perhaps 1–2%, have residual symptoms of low back pain that call for a spinal fusion. Therefore, why do an unnecessary operation with added trauma to the patient and requiring a much longer period of immobilization in hospital?

(*b*) In the event of a re-exploration becoming necessary the difficulties of exposing the theca and nerve roots through the bone graft can well be imagined.

Following operation the patient is usually up on the twenty-first day and out of hospital in four to five weeks. He then proceeds to a rehabilitation centre where he is trained to walk and become normally ambulant. No spinal exercises are allowed. He also wears his Goldthwaite belt from the day he gets out of bed. After twenty-eight days at the rehabilitation centre he then returns to light duties, i.e. about ten to twelve weeks after operation.

The case is reviewed at three-monthly intervals after operation and, provided a satisfactory result has been attained, a full medical category is given at the end of twelve months.

Results

Table I: Number of partial gastrectomies performed in period 1945–1951. Age group 30–50. Relatively fit personnel and good operation risks.

Table II: Gastrectomies—classification of results twelve months or more following operation.

Table I.—Partial Gastrectomy 1945–1951

D.U.	G.U.	Carcinoma	Total
140	52	4	196

Simple Ulcer			
D.U.	G.U.	Total	Died
140	52	192	3

Table II.—Medical Assessment Twelve Months After Operation (1945–1950)

	Simple Ulcer						
	D.U.	G.U.	Total	Died	Full duty	Stomal ulcer	Invalding
Aircrew	18	2	20	—	20	—	—
Ground personnel..	82	26	108	2	103	2	1
Total	100	28	128	2	123	2	1

TABLE III.—LAMINECTOMY (PROLAPSED INTERVERTEBRAL DISC) (1949–1951)

Total	Prolapse	Adherent root	Negative
77	70	5	2

Total	Spinal graft	
77	4	3 for spondylolisthesis at primary operation 1 for residual low back pain after laminectomy

TABLE IV.—PROLAPSED INTERVERTEBRAL DISC

Results of Cases Assessed up to June 1951

Total	Full duty		U.K. and W.E.		Invalided	
	Ground	Aircrew	Ground	Aircrew	Ground	Aircrew
56	48	2	5	0	1	0

TABLE V.—OTHER OPERATIONS

Thyroidectomies		Nephrectomies		Nephrolithotomies	Carcinoma colon	Carcinoma rectum	Sympathectomies		
94		97		36	6	7	99		
Toxic	Simple	T.B.	Hydronephrosis				Cervical	Lumbar	Thoracolumbar
44	50	30	67				20	46	33

If, as a general surgeon, I may be permitted, I should like to refer to the important work done by our orthopædic colleagues in the Service. To mention only a few of the major operations such as menisectomies, treatment of fractures of long bones by the Küntscher nail, and a quite common disability, recurrent dislocation of the shoulder, would merely be to touch the fringe of the work of this department. The majority of such patients are returned to full and active duty on completion of treatment and rehabilitation.

These figures I trust will show that the performance of major operations on Service personnel is well worth while. These individuals are for the greater part cured, as you will have seen, are returned to some form of useful duty and are not lost to the Service.

In conclusion I would like to state that the results you have seen have been in no small part due to the stimulus and invaluable assistance and teaching given to us by our Civilian Consultant in Surgery, Sir Stanford Cade.

Group Captain G. H. Morley, R.A.F.: The Royal Air Force opened a Plastic Surgery Centre of its own at Halton in 1942, under the charge of Wing Commander David Matthews and our Civilian Consultant, Sir Archibald McIndoe.

In 1947, with the completion of treatment of war casualties, this Centre diminished to 25 beds.

I propose to describe briefly the field covered by our activities and to indicate the role of Plastic Surgery in securing high standards of fitness in the Service.

Though we are mostly concerned with the treatment of injuries, defects which are congenital or are acquired otherwise than by trauma also come under our care. To give some idea of the proportion of the chief items, the few figures which I shall quote are taken from one thousand consecutive plastic surgical operations performed since 1947.

The highest standard of fitness after injury is achieved by the completeness of repair and of restoration of function. This is of importance to the Services in peacetime owing to the increasing mechanization of our times. In war, it is of even greater importance.

We treat our *burns* patients from start to finish. Repair may be divided into two phases: (1) Simple grafting operations required to secure healing expeditiously with the minimum of deformity or disfigurement. (2) Reconstructive surgery for the relief of established defects.

Continuity of planned surgery is necessary over many months in most cases, and continuity of experience and study is necessary for the surgeon and his assistants if they are to obtain good results. Though the number of these casualties is small in peacetime, the seriousness of the lesions does not diminish in the cases which occur.

For instance, an airman was burned by petrol which overflowed on to a hot lorry engine on March 10, 1950. This facial burn proved to be almost entirely third degree (Fig. 1). Skin grafts were applied to the forehead, both cheeks and chin on the fourteenth day. The lower eyelids were grafted as an emergency to relieve ectropion on the following day. Later the upper eyelids were grafted and the mouth widened to relieve contracture.

In the next stage, reconstructions of the upper lip, then the lower lip and chin, followed by the nose, were carried out by excision of scar and application of thick dermatome grafts. Eyebrows were provided by hairy Wolfe grafts and the tops of the ears were formed by rolling adjacent skin from the scalp. Meanwhile, his hands had been grafted and a contracted thumb-web was relieved.

This photograph (Fig. 2) was taken a few days ago on his return to duty. At present restricted to the U.K. only, his grafts will settle and I anticipate full fitness in one year.

Skin loss occurs from many causes other than burns. Avulsion of skin is not uncommon, and wide

excision is necessary to secure the adequate removal of some tumours. Repair may be possible from local neighbouring skin, or flaps may have to be brought from a distance.

If it is not possible to undertake the reconstruction immediately, healing should be obtained with free skin grafts or by sewing skin to mucous membrane.

Some years ago a young lady had pneumonia very badly. Pituitrin injections in each arm resulted in extensive and distressing necrosis, in which state I first saw her.

Treatment was excision of the slough, which included all superficial fascia and fat, with immediate cover by free skin grafts—with full functional recovery before the grafts had time to consolidate and become less apparent.

Infection of an exposed raw area, wherever it is or from whatever cause, delays healing. This adds to the depth of granulation tissue and scar, to contractures and deformities. We are fortunate in these days to be able to apply a thin shaving of skin to dress a wound, and to expect almost immediate healing in most cases.

A pilot received a fractured forearm and associated with this injury was a partial glove avulsion of skin from the elbow. The method of repair was by application of sheets of skin graft as an initial dressing. This pilot subsequently returned to full flying duty without restriction.

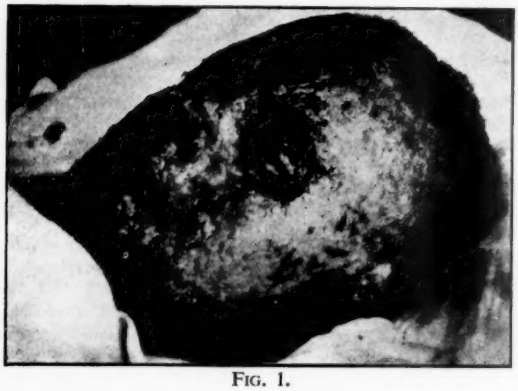

FIG. 1.

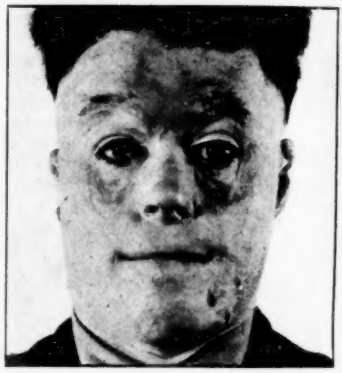

FIG. 2.

Orthopædic surgeons are sometimes hampered in the treatment of fractures where there is an associated loss of skin.

Another pilot suffered a war wound with such a loss from the forearm over the ulna, which was partly blown away. An abdominal skin flap was applied at the first dressing. No other material could heal that wound and cover the ends of the ulna so quickly and so naturally. The defect in the ulna was subsequently repaired and this pilot made a good functional recovery.

Adherent and unstable scar, based upon a thick layer of contracting fibrous tissue and, frequently, with delayed union of the underlying bone, is a barrier through which no orthopædic operation can be attempted.

Local skin flaps of any size are usually unsatisfactory on the leg, so it is often necessary to transfer a living flap of skin from a distance. Infected bone must first be eliminated. Usually the opposite leg is a satisfactory donor. In other cases tubed pedicle grafts are required to bring skin from farther away.

The removal of scar and its replacement with sound skin and subcutaneous tissue heals the wound and relieves the constriction of the circulation both locally and distally. In many cases this suffices to promote union of the fracture; in others, it is the prelude which makes possible the safe surgical exposure and bone grafting of the fracture by an orthopædic colleague.

Ignoring the smaller local flap operations, in this series there appear 31 direct flaps, 15 cross-leg flaps and 9 tubed pedicle grafts, a total of 54 cases (not operations). The standard of fitness in most of these cases is raised from L 7 or 8 to L 1.

The *Physicians* have recently sought our help over recurrent infections of the nape of the neck associated with acne. We have all met these cases. To oblige the defeated dermatologists, we excise the scarred battlefield in its entirety and replace it with a substantial skin graft. We have only done a dozen or so of these cases, but we have an equal number of very grateful patients whose recurrent and painful cause of unfitness has literally been removed.

Fractures of the facial bones vary in situation and in degree, but are characterized by remarkably speedy consolidation.

As with other bone injuries, the principles of treatment are: Early reduction, adequate retention, and preservation of function.

The *mandible* presents special problems which demand close partnership between the plastic surgeon and his dental colleague.

The *maxilla*, if displaced, may need very forcible disimpaction. If reduction is delayed beyond one week it may become extremely difficult. Retention of these fractures can usually be ensured by interdental wiring until dental cap-splints can be prepared.

Severe fractures of the nose are often complicated by concussion or more serious brain injury. The important point is the re-establishment of the nasal airway. Fragments of bone should not be removed unless completely detached.

On July 17, 1949, an officer, a pilot, terminated a motor cycle ride by hitting a fence with his face. Notes from the hospital where he was first treated, read: "Toilet of face, several detached pieces of bone were removed, part of the ethmoid plate deficient, finger could be passed into the anterior cranial fossa through the nose, part of medial wall of the right orbit missing, nasal septum almost entirely deficient."

He came to us three days later with complete collapse of the nose and no nasal airway whatsoever. A severely fractured maxilla and an amputated right leg were complications.

My dental colleague and I operated but could not reduce the nasal bones because there were none The septum was plastered in several fragments on the walls of the nose and had to be sorted ou like a jigsaw puzzle viewed from the side. The airway was restored. The maxilla required no reduction but was in urgent need of retention—it was very loose.

(I should like to take this opportunity to acknowledge the very great debt which we surgeons owe to skilful anæsthetists.)

Eventually, on June 15, 1950, I removed the scars from the nose and lower eyelid, and restored contour with a bone graft.

In May 1951 this officer was categorized at our Central Medical Establishment as A2 G4, Pulheem P6R—for review in twelve months—full fitness for flying duties but restricted to temperate climates because of his prosthesis.

Fractures of the malar may be simple or chaotic. Disability results if collapse of the orbital plate permits displacement of the eye or disturbs the origin of the inferior oblique muscle. Depression of the zygoma may cause trismus and a lateral displacement of the bite of the teeth.

Reduction by the Gillies' method gives dramatic relief in this last condition, but in severe cases the antrum must be opened and its walls restored.

Late restoration of the neglected case can be attempted by the insertion of bone grafts to elevate the cheek and raise the floor of the orbit. The fine adjustment of ocular muscle balance then becomes a matter for our ophthalmic colleagues, who also deal with damage to the naso-lacrimal duct by performing dacryocystorhinostomy.

The importance of these injuries, especially as they not infrequently occur in aircrew, and the tremendous advantage of early and proper repair, can hardly be over-emphasized. I am glad, therefore, to be able to report, from this series, only 40 cases of bone graft to nose, 10 bone grafts to restore the orbital floor and the cheek, 30 nasal refractures and 29 malar reductions.

The hand.—Division of the flexor tendons in the fingers has had an extremely poor prognosis until quite recent years.

In our present series we have 28 operations upon flexor tendons, 13 being tenolysis. 6 were suture of tendons divided in the palm and 9 operations were for the grafting of tendons divided within the flexor sheath. Of these last, two operations were to graft three fingers and in one operation all four fingers were grafted.

Our results are encouraging but, although a very reasonable functioning result has usually been obtained, I must admit to some slight residual limitation of extension, and that it is unusual to obtain the complete range of interphalangeal flexion.

19 operations were undertaken for *repair of damage to nerves.*

There were 13 sutures of the ulnar nerve, 5 of the median, and 1 of both median and ulnar. Mostly, these nerves were divided at, or a few inches above, the wrist. There was also one external peroneal nerve divided at the neck of the fibula.

The finest silk was used as suture material and in this series I have to report only two failures—one in a case which was two years after injury when he came to me for treatment, the other was an un-co-operative apprentice.

In all this work we owe a lot to the sustained enthusiasm and hard work of our Medical Rehabilitation Units. These patients recovered at least 80% of motor and sensory function.

We have treated a healed burn of the forearm and wrist, with a median nerve lesion, by removal of the unstable and adherent scar, which revealed a 3 cm. destruction of the median nerve. An abdominal flap was attached to the forearm.

Later, the median nerve was sutured with a very good result.

Dupuytren's contracture.—This crippling and progressive affliction results in very great disability of the hand.

We have, in the same series, 39 patients involving 49 affected hands, on whom complete removal of the entire palmar aponeurosis has been performed. I stress the need for the excision to be complete, with the relief afforded by the transposition of local flaps on the little finger as advocated by McIndoe.

Of our cases, 12 were officers, 25 airmen, and 2 civilians. Each side was affected about evenly. In 3 cases the thenar eminence was also involved, and in 2 a similar condition was present in the foot.

The results are very encouraging. Further contracture is arrested and there is a considerable relaxation of the affected fingers. If the little finger has been acutely flexed, amputation may be considered, but usually this is left until the result is apparent—in 3 of our cases this was performed later. The two civilians had exceedingly advanced contracture and the little finger was amputated after filleting it out of its skin, which was rotated to relieve the palmar skin defect.

After six months these hands are capable of full function, but until this time has elapsed heavy manual work must be avoided.

Congenital lesions such as the disfiguring cleft lip and the protruding ears, which have become the butt of barrack-room ribaldry, are repaired. The operation does not demand a long stay in hospital and the relief to young men at a sensitive age is enormous.

Hypospadias.—Recently my Assistant, Squadron Leader I. S. P. Wilson, has demonstrated most convincingly that the Denis Browne operation promises reasonable prospect of cure of this most distressing condition. So far, we have treated 12 cases and, of these, only 6 have been completed.

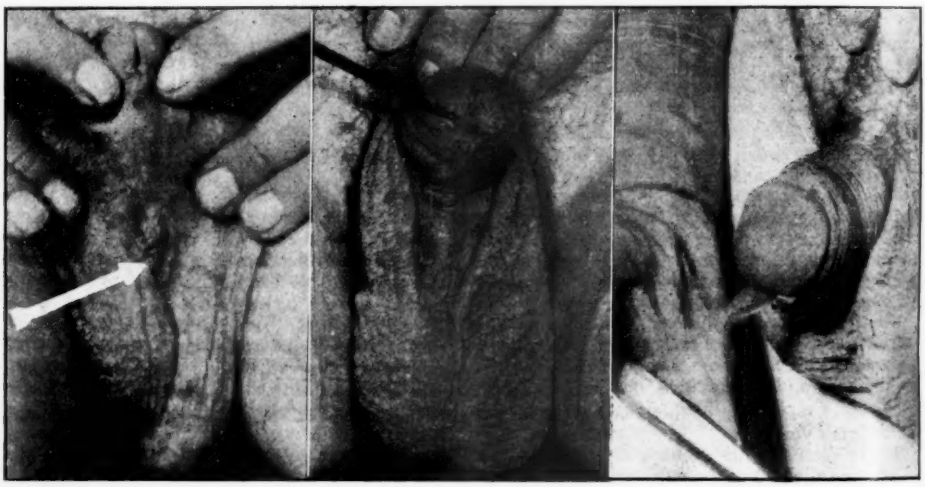

FIG. 3. FIG. 4. FIG. 5.

Satisfactory reconstruction of the penile urethra has been achieved with two periods of one month in hospital, separated by a six months' interval; as occurred in this case (Figs. 3, 4, 5), whose action photograph should be convincing (Fig. 5). I feel that this better prospect of cure justifies the operation in the Service, for I hold that this condition is not only distressing and demoralizing, but that it constitutes a real disability, especially when there is a shortage of pennies!

In concluding, I would suggest that the division of operations into "major" and "minor" only results in the less complex procedures being regarded with disdain. I have never been able to obtain a standard or definition with which to segregate these artificial categories, and I submit that no useful purpose is served to the patient or to surgery by the use of the term "minor".

I should like to acknowledge the steady encouragement and help of Sir Stanford Cade through so many years. In this particular specialty there are hundreds besides myself who owe so much to Sir Harold Gillies, Sir William Kelsey Fry and to Sir Archibald McIndoe.

I also wish to thank the Director-General of Royal Air Force Medical Services for his encouragement and permission to publish this paper.

JOINT MEETING No. 4

Section of Neurology with Section of Psychiatry

Chairman—Professor P. C. P. CLOAKE, M.D., F.R.C.P.

(President of the Section of Neurology)

[*January* 3, 1952]

DISCUSSION ON THE DIFFERENTIAL DIAGNOSIS OF EARLY DEMENTIA

[*Abridged*]

Dr. Martin Roth (Department of Clinical Research, Graylingwell Hospital, Chichester): The mental disorders of old age probably account for the greater part of conditions whose end-result is dementia; they constitute a social and medical problem that, with the progressive ageing of the population in many parts of the world, is likely to advance in magnitude at a rapid pace. Moreover, when dementia occurs early in life, the patient is brought promptly for medical advice, and detailed investigation is usual. In old age, however, there is always the danger that disorders of reversible causation may be assumed to result from one of the irreversible organic degenerations of senility. For these reasons, and because I should like to describe the results of a recent investigation, I shall discuss mainly the senile and pre-senile psychoses.

Most of the classical accounts of the clinical picture of senile psychosis have referred to the depressive symptom-complex as a common feature of the condition. Bleuler, for example, held that recovery from depression in old age was rarely complete, the condition being one stage in the course of a dementing process; this in spite of the fact that Kraepelin had shown that 7·2% of manic depressives developed their first psychosis after the age of 60. Now, as Slater (1951) has shown, using figures obtained by Karagulla from a famous mental hospital, the mortality of untreated depression at all ages varies between 10–14% in females and 16–18% in males. Above the age of 60, deaths from inanition, exhaustion, suicide, and causes unconnected with the psychosis, must add up to a considerably higher mortality. Bleuler's pessimism may therefore have been due in part to the absence in his time of any effective treatment for depressive illness. However, more recently, reports from different parts of the world have indicated that with the use of physical treatments, even patients in their seventies and eighties may at times recover from mental illness. The majority of the patients described had exhibited the depressive symptom-complex. The questions that arose were: (*a*) what was the incidence of such cases in relation to that of the irreversible organic processes? and (*b*) how were they to be distinguished, if indeed they were distinct, from the affective disorders said to result from organic disease of the brain?

A recent investigation at Graylingwell Hospital by Dr. John Morrissey and myself has made an attempt to answer these questions. The results obtained so far suggest that: (*a*) affective disorder, and depression in particular, may be far more common in senescence than has been generally supposed, and (*b*) that its clinical picture and natural history are *essentially* similar to those of affective disorder in earlier life, and that it is a distinct entity sharply demarcated from and probably independent to a large extent of the cerebral degenerations of old age.

A study was made in 1951 of the case-records of all patients over 60 who had been admitted to the hospital during 1948. Total admissions numbered 150, and of these 81 were classified as examples of affective psychosis, 74 (or almost 50% of the total number) being cases of depression, the remaining 7 cases of mania. Affective psychosis was numerically preponderant in each five-year age-group until the age of 75. Above this age, senile psychosis was the largest group, affective psychosis taking second place. There were totals of 36 (24%) cases of senile psychosis, 12 (8%) of schizophrenia, 7 (5%) of arteriosclerotic psychosis, and 14 (9%) of confusion associated with disorders other than those mentioned. It remained to be seen whether the natural history of the different disorders provided any justification for the diagnostic distinctions which had been made between them. It will be evident that only the affective and senile groups contained adequate numbers to offer suggestions that could be considered significant, and most of my remarks will, therefore, be devoted to them. Our classification of cases into these two diagnostic groups had been based on a hypothesis we had formed early in the investigation, that they were distinct disorders; the former with a clearly defined onset and occurring in patients with effective adjustment to the demands of their daily life until immediately before the presenting illness, the latter insidious in onset and presenting with a history of gradually failing adjustment over a considerable period. On the one hand were patients with positive affective symptoms and in some cases delusions in harmony with the affective disturbances. On the other were patients with predominantly negative symptoms of failing memory, deteriorated habits and a variable paranoid colouring seemingly arising from poverty of grasp.

Such study of case-records readily lends itself to subjective judgments. Our follow-up studies, however, provided confirmation for the hypothesis on which our initial classification had been based.

On March 31, 1951, 54 (67%) of cases with affective disorder and 5 (14%) of patients with senile psychosis had been discharged from hospital. The numbers dead in the two groups were 13 (16%) among the former and 28 (78%) among the latter, while in-patients numbered 14 (17%) and 3 (8%) respectively. Further investigation showed that these discrepancies could not be explained in terms of age differences between the two groups. There were 15 patients with affective disorder in the 70–74 year group; 9 of these had been discharged, 3 were dead, 3 in-patients. The figures for cases of senile psychosis were 8 out of 9 dead, and 1 discharged. In the 75–79 year group there were 9 cases of affective disorder; 4 of these had been discharged and 5 were dead: of the 16 cases of senile psychosis, 13 were dead and the remaining 3 in-patients. [1]

A follow-up study of the 70 discharged patients by letter, or visit from a psychiatric social worker, provided us with the following final figures: 53 of the 81 cases of affective disorder maintained a social recovery, while none of the 36 cases of senile psychosis had had a remission; and 89% of the seniles were dead as compared with 23% of the cases of affective disorder. This does not completely describe the benign character of affective disorder, for no less than 61 patients out of the total of 81 had been out of hospital during the 28–40 month period of study for a period of six months or more.

Although the figures suggested that we were dealing with two fairly distinct nosological entities, they could give no precise indication as to the possible degree of overlap between them; but the case records as well as a recent study of 100 consecutive admissions have shown the association between depression and dementia to be sufficiently rare for it to be attributable to the fortuitous coincidence of two relatively common diseases of old age. Follow-up studies have shown, moreover, that a change from depression to dementia or vice versa is very unusual.

A recent psychological and clinical investigation by Miss Barbara Hopkins and myself has provided further confirmation for these views. Of 84 patients over the age of 60 consecutively admitted to Graylingwell Hospital and given tests, 45 were diagnosed as suffering from affective disorder and 15 from senile dementia. (The figures incidentally provided confirmation from current admissions for the predominance of affective disorder.) A battery of tests was administered to every patient. I shall briefly describe some of the results. 36 cases of affective disorder and 9 of senile psychosis were tested with the Progressive Matrices, and a series of questions concerned with orientation, memory and general knowledge was given to 44 patients in the former and 11 in the latter group. On both tests the differences between the two groups were so large and obvious that there was no need to apply statistical tests. The mean score of cases of affective disorder on the Matrices was 18·72 ($\sigma = 7{\cdot}51$), of senile psychotics 2·89 ($\sigma = 3{\cdot}26$; on the Questions the scores were 14·66 ($\sigma = 4{\cdot}17$) and 1·82 ($\sigma = 1{\cdot}89$) respectively. The histograms in Figs. 1 and 2 show the distribution of scores. It will be seen that the groups fall into two distributions that are almost wholly distinct. The slight overlap is produced on each test by a single case of affective disorder. On the Matrices this is a case of mania, so that depression and senile psychosis are in wholly distinct distributions. On the Questions, overlap is due to one case of agitated depression with some confusion, whose score falls into the range otherwise wholly occupied by senile psychosis.

[1] With the numbers in the two small age-groups combined, the difference between senile and affective psychosis in the numbers alive and dead is statistically significant at the 0·1% level.

The test performance of general hospital patients, matched with the senile dements for age, sex and occupational status, shows these differences between the two groups to be independent of differences of age. There was no overlap between seniles and controls on either test; the mean Matrices score for 9 controls was 14.77 ($\sigma = 3{\cdot}74$) and the mean score on the Questions test was 15·90 ($\sigma = 2{\cdot}07$). The results therefore provide confirmation for the evidence in favour of the sharp distinction between the two disorders, obtained from their clinical picture and natural history.

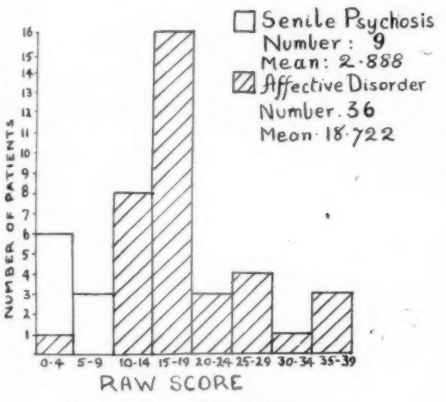

FIG. 1.—Progressive Matrices.

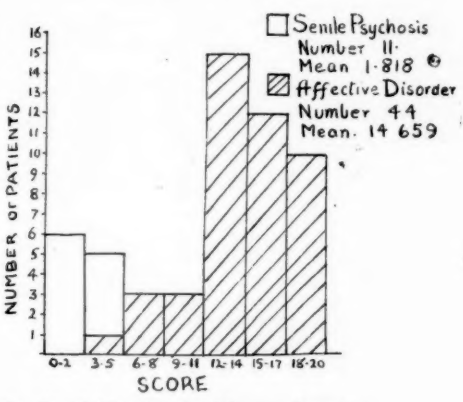

FIG. 2.—Orientation and Memory Questions.

A fifth or more of total admissions to mental hospitals are patients over the age of 60. Hence the practical significance of these findings, if confirmed, is likely to be considerable. Differential diagnosis between depression in old age and senile psychosis is of particular importance, and I have therefore, in Table I, contrasted the clinical features of these two disorders.

TABLE I.—NATURAL HISTORY AND CLINICAL PICTURE OF DEPRESSION IN OLD AGE AND SENILE PSYCHOSIS (BASED ON 110 CASES OVER 60 YEARS ADMITTED IN 1948)

	Depression in old age (74 Patients)	Senile psychosis (36 Patients)
Family history of mental disease	In 45%	In 17%
Past history	Depressive illness before 60 in 42%	Various mental disorders before 60 in 8%
Onset	Rapid	Insidious
Attempted suicide	Common	Rare
Affect	Depressed	Euphoric, fatuous
Thought	Coherent	Incoherent
Delusions	Systematized, in harmony with affect, concerned with guilt, punishment, nihilism	Unsystematized, ever-changing, arise from lack of grasp
Orientation	Correct	Deranged
Confusion	Rare	Common
Course	Remitting	Progressive
Outcome after 28–40 months	53% social recoveries 23% dead	No recoveries 89% dead
Effect of E.C.T.	Remission or recovery	Course unaffected
Performance in psychological tests	Within normal range for age	Well below normal range for age

Arteriosclerotic psychosis.—The status of this condition has recently come into dispute. Doubt has been thrown upon the relationship between cerebral arteriosclerosis and ageing in general (Hirsch, 1945; Riese, 1946). The severity of the clinical picture has been shown to bear no relationship to the extent of the changes in the brain (Rothschild, 1937, 1942, 1945). It has also been widely

claimed that a differential diagnosis between senile and arteriosclerotic psychosis is generally impossible and is, in any case, of little practical value. None the less, whatever their underlying pathology, cases with patchy impairment of intellect and personality, fluctuating course, relatively good insight, often with focal cerebral signs and symptoms and emotional incontinence, form a very distinctive group. There were 7 (5%) such cases in our 1948 material. They tended to remit more often than senile psychotics, and psychologically the few cases we have tested so far have shown a better retention of intellectual capacity. As many cases commence before 60, the figure of 5% does not reflect the true incidence of the condition even in mental hospitals. Even so, I believe that strictly defined, it is not a common disease. The lower age limit of 60 years may also explain the absence from our material of cases of pre-senile dementia and general paralysis of the insane.

Confusion.—This is a basic problem in relation to dementia in old and middle age. It may be merely an episode in the course of an organic psychosis, but it also occurs in the old as a sudden and unexpected complication of acute infection or following operations, particularly those for prostatectomy and removal of cataract. It is always a serious complication, which takes an increasing toll as age progresses, from rapid exhaustion, starvation and intercurrent infection. However, when it develops acutely in patients with well-preserved personality and intellect who are not frail, complete recovery may occur.

In our 1948 case-material there were 49 patients with confusion. Six months after admission to hospital, 18 of the 28 cases of senile psychosis confused on admission were dead. Of 5 cases of confusion complicating acute or extensive physical disease, 3 were dead within a week, 4 in a month, all in six months. In these conditions then, confusion is a grave complication, and in very ill or feeble patients likely to prove a terminal one. The 3 patients whose depression was complicated by confusion were likewise dead; they were aged 68, 79, 75, and 2 of them died of physical illness whose association with the mental disorder may have been fortuitous. In 5 cases, confusion had suddenly developed for no clearly-defined cause. 3 of them were discharged, and follow-up studies have shown them all to be well. The prognosis in such "non-specific" cases is thus relatively good.

The sequel to an acute confusion is frequently the amnestic (or *Korsakoff*) syndrome. It is not an inevitable sequel, nor is it always preceded by confusion. It is a non-specific preformed cerebral response which may occur in association with alcoholism, senile dementia, general paralysis, arteriosclerosis, tumour or head injury. If a systematic examination is not carried out it is easily missed, for superficially the patients seem cheerful and alert and may conduct an apparently intelligent conversation for short periods. The reason is that their defect may be remarkably focal. Careful examination reveals it as an inability to retain recent impressions for longer than minutes or even seconds—even the fact that he is hemiplegic may not be remembered by such a patient. However, in most cases all aspects of memory are involved—attention, grasp, retention and recall, as well as ordering of the temporal sequence of events. A fatuous euphoric affect, lack of spontaneity and suggestibility, are constant features. Confabulation varies with the degree of impairment of initiative. If their attention is kept stimulated, some patients with the amnestic syndrome nevertheless perform remarkably well in intelligence tests, and unless memory is included as a criterion, such tests will fail to demonstrate deterioration.

Continental authors such as Gamper have claimed that a basal diencephalic lesion is adequate by itself to cause the amnestic syndrome, and there is much evidence to suggest that this is at any rate one way of producing the condition. Bleuler (1951) has, however, argued recently that it is indicative of chronic diffuse cerebral damage. But the chronic cerebral tumours from which he derived his conclusion do not provide a sound basis for choosing between theories of diffuse and focal determination, since pressure on the diencephalon may have been the factor common to all the tumours in different situations studied in his Clinic. Apart from such problems in cerebral localization, there are many clinical and psychological problems urgently awaiting solution. For this is a condition in which facility of emotion and decreased volition are in some way inextricably bound up with an incapacity by the brain to form new and enduring engrams. It is therefore a fascinating problem of the most fundamental importance, and its investigation from many angles may be expected to throw a much-needed light on the subject of dementia, for the extent of our knowledge is here greatly overshadowed by the level of our ignorance.

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Dr. John D. Spillane (Cardiff): Progressive loss of brain power or dementia may be the outcome of long-standing insanity or it may be the first evidence of structural changes in the brain. Classification of the dementias must at present be essentially practical. As Hughlings Jackson once said: "It would be as absurd to attempt to arrange patients in an asylum on the principle of dissolution as it would be to arrange plants in a kitchen garden on the 'natural system' of botanists."

I shall discuss only the purely clinical aspects of some of the dementias of organic origin in adult life.

Before considering the causes of dementia let us first be sure that the phenomena which suggest the diagnosis are in fact pathological. Secondly, let us see how progressive intellectual failure may be mimicked or masked.

Perhaps the term "dementia", like that of "euphoria", is too readily used nowadays. There should be adequate reasons for making the diagnosis and allowance must be made for the wide variation in mental processes in middle-aged and elderly people who comprise the majority of patients. It is curious how symptoms such as irascibility, forgetfulness and garrulity in the elderly are often assumed to indicate cerebral deterioration while the same phenomena in the young pass unnoticed. Little is known of the effect of ageing on the results of standard mental tests and the clinician's estimate of prognosis should not be unduly influenced by results which are not in accord with clinical evidence.

If, then, dementia must be distinguished from mere ageing neither must it be confused with conditions which, although affecting mental capacity, are often temporary. A difficult differential diagnosis may be that between early organic dementia and an involutional depression. The manner in which mental performance may improve after a course of electrically induced convulsions is sometimes quite striking. In this type of case nothing is more important than the clinical judgment possessed by the clinician—there may be no signs or ancillary evidence to aid him. Then, too, a toxic confusional state may present as an episode not unlike that which punctuates the course of arteriosclerotic dementia. Uræmia must always be watched for—especially that variety in which headache, insomnia and apathy dominate the picture.

The diffuse impairment of the mental functions which characterizes dementia must be distinguished from the effects of specific disabilities arising from focal cerebral lesions. "Forgetfulness" may be the first sign of nominal or amnesic aphasia, while what was assumed to be deafness might prove to be auditory agnosia. A visual hallucination in an elderly person might only mean a lesion in one retina. "Wandering" or losing the way home may result from the visual agnosia of a parieto-occipital lesion. Apractic phenomena may be misinterpreted as evidence of deterioration of a diffiuse character. Disorientation, subtle enough to escape routine examination may explain remarks and behaviour falsely attributed to dementia.

On the other hand a considerable degree of camouflage of failing faculties is possible. Conversation which is apparently fluent and normal may merely be a continuation of remarks already voiced. An inability to embark on a conversation as opposed to drifting with one was the first thing to arouse the suspicion of a wife that something was wrong with her husband. In a man of obsessional habits, serious failure of memory was hidden from a colleague for several years by reliance on careful organization of daily activities. In some early dements showing the frontal syndrome, the seeming rapidity of thought, the promptness of reply, the assurance of manner and the ease with which decisions are made may completely deceive for a time.

It is generally true to say that the early signs of dementia vary little whatever the underlying pathology, although the course of the illness will naturally differ. Either in thought and memory or in feeling and conduct a change takes place. When this underlines and accentuates previous personality traits, then realization of what is going on will be delayed. Perhaps the patient with cerebral arteriosclerosis was always inclined to be depressed or paranoid in his outlook, or the patient with a frontal lobe tumour or general paresis was always somewhat aggressive and boastful. The balance achieved in the course of a lifetime by the activity of inhibitory influences—both voluntary and imposed—is now in danger. Integration is failing. When something foreign to the person's nature makes its appearance there is less difficulty. Probably a balanced and well-integrated character will withstand the influences of disintegration of his mind longer than would he whose outward behaviour was never a true reflection of personality. The anonymous author of a poignant article entitled "The Death of a Mind" which appeared in the *Lancet* last year (1950, i, 1012) traced the progress of dementia in her brilliant father. "The change," she wrote, "was so slow as to be hardly perceptible and the signs vanished when I tried to pin them down; they were like those faint stars which are seen more easily when they are not in the direct line of vision. . . ."

Differential diagnosis.—Firstly, the clinical history may disclose a previous attack of acute encephalitis or serious head injury. There may be long-standing epilepsy or evidence of chronic alcoholism or familial dementia. Occasionally some unusual story is obtained. In a recent case dementia followed a thoracic operation during which there was cardiac arrest for six minutes. Dementia may follow carbon monoxide poisoning or irradiation of the brain.

Secondly, there is a group of cases in which the cause of the dementia may be suspected when the patient is seen. There is the well-known appearance of the patient in myxœdema, in Cushing's syndrome and in chronic alcoholism. The involuntary movements of Huntington's chorea or the ataxic movements and speech of cerebellar atrophy may attract notice. Sudden weeping or laughter during the interview may indicate the arteriosclerotic basis of the dementia.

Thirdly, the cause of the dementia may not be far to seek in that routine investigation discloses such evidence as papillœdema, a positive Wassermann reaction in the cerebrospinal fluid or internal frontal hyperostosis.

Head injury.—Dementia that has not appeared within two years of the injury is unlikely to be post-traumatic in origin. Or, again, the original injury was not perhaps followed by any definite period of confusion and amnesia. In the elderly, of course, the injury may have been to a brain already affected by arteriosclerosis or atrophy. It may aggravate and hasten any decline. The alcoholic patient, too, is more liable to injury and he may then present a picture not unlike that seen in the Wernicke and Korsakoff syndromes. A history of injury then must always be scrutinized carefully. Although the previous personality is of acknowledged importance in determining the rate and degree of recovery from head injuries, as a whole, it has not been shown that it is clearly related to the problem of post-traumatic dementia.

Cerebral tumour in the elderly often causes no increase of intracranial pressure, presumably because cerebral atrophy allows of greater accumulation of cerebrospinal fluid. If the illness is ushered in with an acute cerebral episode and papillœdema does not develop then tumour may not be suspected. Indeed it is sometimes impossible in such cases to decide on purely clinical grounds whether the cerebral lesion is necrotic or glioblastomatous. A slowly progressive hemiplegia with increased cerebrospinal fluid protein accompanying the mental deterioration is commonly due to glioblastoma.

Transient cerebral episodes, especially if separated by intervals of some months' duration, point more to cerebrovascular disease. Insight is then retained and symptoms fluctuate. The patient with cerebrovascular disease is often full of complaints—somatic and nervous—insomnia, bad dreams, indigestion, loss of weight and appetite and bizarre pains in the head and face. In such cases the cerebral changes may so alter the personality of the patient that neurosis develops for the first time. Giddiness, syncopal or epileptic attacks may occur while transient cerebral palsies will indicate the vascular basis of the illness. Sclerosis of the radial and retinal arteries is not a good guide to the state of the intracerebral arteries. Retinal arteriosclerosis seems to correlate more with changes in the basilar artery.

Cerebral tumour.—There is no mental disturbance peculiar to cerebral tumour. The agent responsible for the production of, say, a frontal syndrome is not identified by the mere existence of certain neuropsychiatric phenomena. That it is the spirochæte or alcohol or tumour is suggested by the presence of other changes and by the course of the illness. Indeed experience has shown that lesions of the temporal lobe are almost as likely to cause a so-called frontal syndrome. As for their value in the localization of tumours, there is no comparison between the significance of such specific disturbances as aphasia, apraxia and agnosia and that of general psychopathological syndromes. Dementia is here on a par with epilepsy as a manifestation of cerebral tumour. It has little localizing value.

When there is intracranial hypertension there is likely to be progressive impairment of awareness with perhaps episodes of confusion. Such signs as indistinctness of speech, mild facial weakness, slowing of the pulse-rate and loss of the abdominal reflexes may provide clues to the nature of the illness. The torpor of raised intracranial pressure should not be confused with the hypersomnia of hypothalamic lesions or with the more florid changes in personality which tend to take place when a tumour involves the frontal or temporal lobes, the corpus callosum, the thalamus or third ventricle. Tumours so placed not infrequently produce striking alterations of personality and behaviour which raised intracranial pressure of itself rarely does. The intellectual deterioration, disturbance of affect and memory may be identical in frontal and temporal tumours. When there is involvement of the thalamus and walls of the third ventricle, emotional instability and degeneration of character can be pronounced.

Carcinomatous infiltration of the meninges may run a surprisingly slow course characterized by increasing dementia. A fall in the chloride content of the cerebrospinal fluid may be noted.

Of subdural hæmatoma I would say only this. Firstly, not only may the cerebral compression result in progressive impairment of intellect and memory but it may also be the cause of a sudden psychotic episode. Secondly, it may evade detention even after burr holes have been made as in the case where it was only the instinctive demand of a wife for an autopsy on her late husband which led to the disclosure of a chronic encapsulated hæmatoma. She wanted to be able to reassure her children that their father's dementia was not genetically determined.

Mr. M. B. Shapiro (Department of Psychology, Institute of Psychiatry, London):

Use of Psychological Tests for the Differential Diagnosis of Early Dementia

It was just over twenty-one years ago that Babcock (1930) published her first article in which she demonstrated a new method for the measurement of dementia. She showed that a discrepancy between the vocabulary test on the one hand and certain non-vocabulary tests on the other differentiated significantly between a group of paretics and normals. This laid the basis for the use of discrepancies between cognitive tests as measures of dementia, such as the Wechsler Index of Deterioration and the Shipley-Hartford Scale.

This method, and its derivatives, are fast reaching an impasse. The first reason for this is that where they provide statistically significant differences between normal and abnormal groups the overlap is always too great to be of clinical use (Botwinnick and Birren, 1951). Secondly, where an attempt has been made to differentiate between brain-damaged and other psychiatric groups (Rogers, 1950) no significant differences are demonstrable.

These difficulties may be due to the fact that the Babcock technique, in its various forms, tries to do three different things. First of all it aims to measure the discrepancy between past and present levels of intellect. The vocabulary test is used as a measure of the past level and the non-vocabulary tests as a measure of the present level. Secondly, it aims at comparing the patient's present intellectual level under unspeeded conditions with his level under speeded conditions. Vocabulary now becomes the tool to measure the first, and the non-vocabulary tests the means of measuring the second. Finally, it aims to measure previous memory compared with present memory. Here vocabulary is called upon to measure the first, and a selection of non-vocabulary tests the second.

A priori there is no reason why a single index can be expected to fulfil these three different functions. In fact, there is now every reason to expect that any one of these three types of discrepancies, being possibly the outcome of complex processes, may need to be measured by more than one kind of tool, and that the Babcock index itself is a complex instrument, measuring a number of very different functions in as yet unknown combinations. For example, we know that vocabulary does have a high correlation with a general intellectual factor. In addition it is less affected by brain damage than other intellectual functions, and is, in the form used by Babcock, given as an unspeeded task. Again, Babcock's wide variety of non-vocabulary subtests would tend to cancel out the effect of additional broad and specific factors and thus result in some kind of pooled estimate of "G" as defined by Spearman (1932). The non-vocabulary part of the test could therefore serve as some kind of a "G" test which was speeded and at the same time relatively free of the influence of past learning and past level of "G". Thus it is possible for the Babcock type of index to serve, because it is complex, some of the demands made of it and, precisely because of this, serve each of them with relative inefficiency.

In the light of this analysis it appears to be necessary to discard Babcock's and other similar indices in their current forms, and to reformulate and to test experimentally Babcock's and other theories of the psychological outcome of different types of disorder. This would necessitate the development of new tools such as Furneaux's speed and power test, and clearing up the problem presented by the fact that current clinical memory tests appear only to test "G" (Eysenck and Halstead, 1945).

I will now turn from the discussion of the quantitative to a consideration of the qualitative methods of measuring dementia. Recent years have also seen considerable development in the popularity of the so-called "qualitative" methods, among the most prominent being those developed by Goldstein and Scheerer (1941). Aside from the lack of sufficient validation data, the possible diagnostic usefulness of these tests in their present form is further diminished by the claim that abnormalities are shown in subjects suffering from functional disorders and amentia as well as brain damage, thus creating an impasse almost identical with the one we found in the quantitative methods of measuring dementia.

These difficulties led to the writer's (Shapiro, 1951) development of an experimental investigation of the dynamics of one of the anomalies observed in the Goldstein-Scheerer Block Design test: the rotation effect. This consists of copying the design correctly with four blocks but placing them in an orientation which is rotated to an angle of at least 15 degrees and often as much as 45 degrees from the orientation of the original design. The stimulus conditions which maximize and minimize the appearance of the rotation effect have been established and they have been found to differentiate significantly between brain-damaged and functional patients, none of whom was over 45 years of age or defective in intelligence, with a misclassification of about 25% (Shapiro, 1952). Furthermore, Goldstein's theory that the rotation effect is associated with subcortical lesions is confirmed by a recent testing of 9 Parkinsonians, 8 of whom showed this effect to a marked degree. At the same time, explanations of these findings have been developed which have significance for the general behaviour of brain-damaged patients, and which are now being put to experimental test.

Another qualitative test which is popular is the Rorschach. The outstanding finding with this test is that of Hughes (1950) who reports a powerful differentiation between his functional and

brain-damaged patients. He established an index which would pick out 82% of his 50 organic patients and only include 1% of the remaining 142 patients. This finding is the best yet reported; it does not apply, however, to elderly or dull patients.

Perhaps the most interesting finding is that of Werner and Thuma (1942). They found that dull and retarded brain-damaged children were unable to see apparent motion, contrary to all but one of a matched non-brain-damaged group.

It may well be that the more elementary perceptual functions such as flicker fusion and apparent movement will turn out to be very sensitive to pathological conditions in the brain. This expectation is confirmed in the report by Bender and Teuber (1947) of a patient with a known parieto-occipital injury due to a gun-shot wound, and in whom it was difficult to elicit defects by conventional methods.

It is clear from this paper that the psychologist is not in a position to place in the hands of the psychiatrists validated tools for the diagnosis of early dementia. For practical purposes, however, a number of tests might be used as an aid to clinical judgment. These are Brody's shortened version of the Babcock Scale (Brody, 1942), the full-scale Wechsler I.Q., Werner and Thuma's apparent motion test, and Hughes' Rorschach index. Each of these instruments could, of course, only be used for patients who are comparable with the samples on whom the tests were originally validated.

There are many unvalidated tests available, but such tests are a handicap rather than an aid to the psychologist. They become a barrier between himself and the properly controlled observation of his patient.

The paramount need appears to be a systematic application of experimental method to the solution of precisely formulated problems. In my opinion the immediate aim of such investigations should be not so much to find new diagnostic tests, but rather to seek explanations of the phenomena as we observe them in our patients. Such explanations, tested by controlled experiment, seem to me to be more likely to place sensitive tools in our hands.

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Section of General Practice

President—J. D. SIMPSON, M.D., M.R.C.P.

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DISCUSSION ON THE POSSIBLE FUTURE USE OF CORTISONE AND ACTH IN GENERAL PRACTICE

Dr. Talbot Rogers in opening the discussion on the possible future use in general practice of cortisone and ACTH explained that he had drawn his conclusions chiefly from the experience he had shared at Lewisham Hospital, where a research unit under the direction of Dr. Allott was entrusted with the whole of the supplies of these two drugs at present available in the South-East Metropolitan Region. Practitioners naturally wanted to know what were the chances of their having these drugs available for use at their own discretion in their own practices. At present supplies were so limited and the cost so high (the average cost of the maintenance of treatment on minimal effective doses still lay between £10 and £20 per patient per month) that for a long time to come, what was available must remain in the hospitals and even there be used chiefly as a research instrument, or for the treatment of dangerous illnesses for which no alternative effective treatment was known. Nevertheless the general practitioner should be kept aware of the experience of those who were working in the hospitals with these drugs. He should know of the potentialities and the limitations of the drugs. He could play a most important part in the detection and selection of cases suitable for treatment on clinical and on social grounds. He also had his part to play in supervising the maintenance of treatment after his patient had left hospital, and particularly in the assessment of clinical progress and the detection of side-effects.

Following a brief description of the adrenal cortical hormones, and of the effects of ACTH upon the adrenal cortex, Dr. Rogers went on to list the illnesses for which treatment by cortisone or ACTH had been found effective. Chief among these were rheumatoid arthritis and Still's disease (ankylosing spondylitis seemed less amenable to treatment). The results in acute rheumatic fever were not yet so clear but Kendall claimed that "In the acute phase of rheumatic fever, cortisone will abolish the symptoms, and it seems to protect the heart from damage". Considerable success had been obtained in the treatment of status asthmaticus, in acute lupus erythematosus, and in several types of inflammatory diseases of the eye. Some skin diseases such as exfoliative dermatitis, acute psoriasis, and pemphigus responded well. The drugs had also been effectively used in the immediate treatment of severe burns.

The drugs tended to delay healing and they were therefore to be avoided in patients suffering from active or recently healed tuberculosis, or from active peptic ulcer. Septic conditions often became rapidly worse when treatment by cortisone or ACTH was commenced. ACTH sometimes upset the sodium, chloride, and water balance, and was therefore contra-indicated in hypertension and congestive heart failure. It might also induce hyperglycæmia and was therefore to be avoided in cases of severe diabetes.

Undesirable side-effects were not uncommon during the administration of the drugs. They included œdema, hypertension, glycosuria, fatigue (associated with low potassium level in the blood) psychic disturbances—such as exaggerated euphoria, and insomnia. Prolonged administration could also result in the development of the Cushing syndrome (hirsutism, moon-face, acne, amenorrhœa, obesity over pectoral, abdominal, and upper dorsal areas; the nutrition of the skin suffers, striæ appear, and sometimes ecchymoses). Most of these side-effects did not persist after treatment was given up, but as the original disease was likely to return on abandonment of treatment, a decision had sometimes to be made between allowing the persistence (or even the worsening) of side-effects, or risking the sudden and severe return of symptoms on withdrawal of treatment. These withdrawal effects were likely to be even more marked on the cessation of cortisone therapy than on the withdrawal of ACTH. Apparently the effect of regular administration of cortisone was to make the adrenal cortex lazy, and materially to reduce the normal production of cortisone.

It has therefore become obvious that the sudden cessation of treatment is usually undesirable, and sometimes dangerous, and most hospitals using the drugs now aim at investigating cases to see whether they are suitable for treatment, finding first the optimal dose for the elimination of symptoms, and then gradually reducing the dosage until the minimal maintenance dose is found, which, although not banishing symptoms, keeps the illness under control, and allows reasonable activity for the patient. Most of this assessment depends upon clinical observation. Laboratory tests have not been very helpful in determining progress or in estimating prognosis. The E.S.R. does fall on full doses, but with minimal maintenance doses often remains disappointingly high. Other laboratory tests such as the excretion of 17-ketosteroids and the Thorn test had proved their value in the original investigation, but were not necessary during the period of maintenance therapy. Probably the most useful investigation for the practitioner to carry out during this maintenance period was a check upon blood pressure and body weight (which latter rises quickly if water retention is occurring).

ACTH needs to be given by injection, intramuscularly or intravenously, at regular and short intervals. One of the most economical (but least practical) ways of giving the drug is by continuous intravenous drip over twelve to twenty-four hours. Given intramuscularly three doses in twenty-four hours are probably needed if satisfactory results are to be obtained (certainly not less than two). Cortisone can be given at longer intervals. Many patients are being kept satisfactorily controlled on doses every other or every third day. Parenteral administration of cortisone is now being extensively used in the U.S.A., less so in this country. Its use demands the intelligent and determined co-operation of the patient who must understand the importance of regularity and exactitude of dosage, and the folly of any sudden cessation of treatment—be this due to disappointment with progress, or to economic inability to continue treatment indefinitely.

If these factors are realized, there is no doubt a growing field for co-operation between hospital, practitioner, and patient in the initiation and maintenance of treatment by ACTH and cortisone, and it is not improbable, as the months go by, that the simultaneous use of these drugs, and of the more traditional methods of treatment by gold therapy, and the various forms of physiotherapy, will be more widely developed, and that practitioners will find an increasing possibility of helping some of their rheumatoid patients to live more comfortable and more active lives.

Dr. W. S. C. Copeman pointed out that cortisone and ACTH represented an entirely unprecedented type of therapeutic agent, since they are not in any way curative of disease processes but apparently act by protecting the vulnerable tissues at cellular level from the effects of the disease. For this reason the patient can be expected to revert to his original state on cessation of hormone therapy of this type, and generally does so. Clinical experience regarding the best and safest methods of using these substances is still comparatively limited, and it is the opinion of many that the available supplies might best be used in the experimental field for some time to come. Since, however, both cortisone and ACTH are, to some extent, already being used as routine treatment, it is important that the general practitioner shall have some theoretical knowledge of the indications and contra-indications for their use, and the methods of dosage.

He pointed out that the aim is to find a method whereby patients can be maintained on these hormones over long periods without the risk of producing undesirable side-effects. As this aim has not yet been achieved, busy general practitioners who are not prepared carefully to keep their patients under frequent, regular observation, were advised to avoid such therapy until it is better understood.

Since these substances will not reverse changes which have become permanent, proper selection of patients is important. Contra-indications include hyperpiesis, any tendency to cardiac failure, a history of psychoneurosis, or diabetes in the family, renal impairment, gastric ulceration and pyogenic or tuberculous infection.

The preliminary assessment of the patient chosen for such therapy should take place in hospital in order that a base-line may be established at the onset from which any later suspected deviation may be measured. The maintenance dose suitable for each patient can also best be arrived at during this period. When the patient returns home the practitioner has the choice of administering cortisone by intramuscular injection or by mouth; the dosage is similar. ACTH can only be given by injection and is at present inconveniently short-acting. The average patient requires 50–75 mg. cortisone once daily to maintain him in reasonable comfort at a safe level. It is found that the maintenance needs of patients often vary from time to time, and "booster" doses may sometimes become necessary for a few days. When dosage has to be cut down or stopped, this must be done very gradually in step-like fashion in order to avoid the "withdrawal" syndrome which will otherwise occur.

Dr. Copeman mentioned the chief side-effects which may occur and stated that these may prove sufficiently severe to necessitate cessation of treatment. In an exceptionally carefully controlled series, which he mentioned, however, it had proved possible to return 17 of 20 patients to full employment, and keep them there. One patient only had gone into complete remission, and this had persisted for eight months so far. Side-effects had not been troublesome in this series, no doubt on account of the almost daily observation which had been maintained throughout.

He thought that supplies of cortisone and ACTH would be likely to increase in the near future, although he thought it doubtful whether the price of these hormones would fall substantially owing to the difficulties of manufacture.

He then showed a short cinematograph film to illustrate the effects of treatment with cortisone.

Dr. E. N. Allott said that although he would stress the laboratory side of the problem, he would base his remarks on the experience obtained during the last year when the Regional supplies of cortisone and ACTH were being used at Lewisham Hospital. He agreed with the previous speakers, that the present position is an experimental one and the treatment cannot in any way be regarded as established routine.

The majority of cases for which cortisone and ACTH are of value seem to require prolonged therapy and the problem of future maintenance had limited very seriously the number of cases it had been possible to investigate and treat. The conditions in which the greatest success was obtained, apart from rheumatoid arthritis, were proved cases of disseminated lupus erythematosus, where the drug apparently was life-saving, but maintenance had, so far, proved necessary. A number of comparatively rare conditions had also been investigated but the results were not sufficiently definite at present to be discussed in detail. Promising results have been obtained in some cases of severe burns.

In general it was found that ACTH was more rapid in action than cortisone, but appeared to be more variable and perhaps rather less reliable. Most of the patients felt better on cortisone than on ACTH and the fact that ACTH injections have to be given at least four times a day in most cases limits its usefulness for maintenance purposes. A long-acting ACTH is now available in America but not as yet in this country. Large single doses of ACTH have very little effect. We have recently had supplies of cortisone tablets which can be given by mouth, but have not had experience of using them up to the present. Apparently the effect of cortisone is much more rapid when given this way than by the subcutaneous route. The most efficient way at the moment that we have found of using ACTH is by slow intravenous infusion and the longer this lasts, the better. A dose of about 30 mg. over twenty-four hours will produce the maximum effects.

Most of the laboratory investigations, which are very interesting in helping to understand what is happening and which are useful and necessary in special investigative work, are not of very great value in the control of treatment, and for this purpose they seem to be unnecessary. It is possible to get considerable clinical improvement even while the E.S.R. is still raised. The most important thing in the control of treatment is the patient's clinical condition: undesired effects such as retention of fluid, rise in blood pressure and any symptoms of potassium deficiency, such as weakness, must be watched for carefully. The danger of lighting up quiescent tuberculous infection should also be borne in mind.

The financial factor limits the use of these drugs at present. Neglecting the cost of treatment in hospital, which may be very considerable, the smallest dose of cortisone which can be used for maintenance, as far as can be seen, for an indefinite period, is something of the order of 25–30 mg. a day; this is sufficient to restore to working efficiency only a comparatively small proportion of patients; many should have three times as much. At present prices the minimum cost is something of the order of £70 a year and the cost for adequate treatment is more commonly a figure approaching £200 a year. On account of the complicated chemical processes involved in its manufacture, it seems unlikely that the price of cortisone will ever fall to a low one; although it may come down considerably from these figures, it will still be a very expensive form of therapy. ACTH is about five times as expensive, gramme for gramme, but as the dosage is probably only about half that of cortisone, the actual cost for treatment is about twice. However, as previously mentioned, it cannot at present be used conveniently for maintenance.

Dr. D. K. Briggs said that an idea of the possible future general use of cortisone and ACTH was to be gained from the study of the work done in a hospital where the hormones were freely available, but choice of cases was limited.

Nine cases of rheumatoid arthritis responded well; cortisone produced uniform results but ACTH was less reliable and in one case made the disease worse. A patient with Reiter's syndrome was successfully protected from the effects of the disease for a period of three months until natural remission supervened. Maintenance treatment was at all times easy with cortisone but difficult with ACTH.

Foreknowledge of chances of success in a given case could be gained from the clinical response to the first ten days of treatment. If the sedimentation rate and eosinophil count fell during the early days of treatment, then these estimations would continue to serve as useful guides.

A patient with ulcerative colitis and several with acute skin diseases appeared to respond well to ACTH but interpretation of isolated results was impossible.

ACTH was successful in 6 out of 7 cases of asthma but uniformly ineffective in pulmonary fibrosis. A desperate attempt to relieve thrombo-angiitis obliterans in a young man in danger of amputation of his remaining leg failed. In 2 cases of Hodgkin's disease, there was improvement in the general condition in one.

A case of idiopathic acquired hæmolytic anæmia appeared to make a dramatic response, but later relapsed and died in spite of large doses of both hormones.

Laboratory control was essential, as instanced by the sudden, unexpected appearance of sodium deficiency in one case; in general, however, indications for laboratory investigations did not differ from those existing in other branches of clinical medicine.

Dr. Kenneth Robertson (Southampton) confirmed the experience of other speakers regarding the value of cortisone in status asthmaticus. He stressed that not only were the severe symptoms frequently resolved, but that following treatment with cortisone there was not infrequently a considerable remission of the asthmatic condition. He also described an acute allergic attack in a colleague, following the experimental injection of a staphylococcal antigen. The symptoms having failed to respond to antihistamine drugs, resolved dramatically after two doses of cortisone by mouth. A most severe and resistant case of stomal ulceration responded immediately to ACTH given in an 8-hour drip, and a case of chronic lymphatic leukæmia showed rapid reduction in the size of enlarged lymph nodes and spleen, together with improvement in the patient's general condition though the blood picture was unaffected.

Dr. H. Stephen Pasmore asked for further information concerning the uses of cortisone in diseases of the eye, and mentioned the case of a patient with severe iridocyclitis with K.P. following herpes zoster and later complicated by secondary glaucoma. After eight months' treatment this patient's life was still being made miserable by the acute discomfort and constant lacrimation of the eye. Cortisone drops were then instilled with rapid disappearance of these two troublesome symptoms.

[*January* 16, 1952]

DISCUSSION ON INFANTILE PARALYSIS

Dr. Donald Cameron: The aspect of poliomyelitis which concerns the general practitioner is principally the clinical picture, and it is this aspect which I intend to discuss.

Poliomyelitis is due to a virus which can be isolated from the nasopharynx, intestinal tract, and post mortem from the central nervous system. The incubation period is thought to be ten to fourteen days though it is said that it may be as short as three or as prolonged as thirty days or more. In earlier times poliomyelitis was regarded as a disease of infants. Nowadays, in this country, it affects both the young and old. If not the very old, certainly people of all ages up to their fifties. I have gone through the records of 100 consecutive cases occurring in East Anglia which have passed through my hands since 1947. Most of these occurred in the epidemics of 1947 and 1949. Males were affected more than females in the ratio of 62 males to 38 females.

The Age Incidence

In the age group 0–14 there occurred 48% and in the age group 15–50 42% occurred.

It seems clear, therefore, that the term "infantile" as applied to this much-dreaded disease is not strictly correct. It may be, of course, that there has been a slide in its incidence towards a higher age group than was the case earlier.

The Seasonal Incidence

Hitherto one had always regarded polio as occurring during the hot, dry months of the year but in this group of cases the month of highest incidence in 1947 was September, in 1949 the highest incidence was October and in 1951, our present winter, the worst month was November.

Clinical Observations

It is generally accepted that a person may harbour the virus of poliomyelitis without showing any clinical evidence of the disease, and it is thought that the carrier-rate is very high, it being estimated that the number of people who become immunized may be in as high a proportion as 100 to 1, meaning that for every case notified, one hundred people may acquire immunity, having possibly had subclinical forms of infection.

One finds, in a certain percentage of cases, a history of a minor illness, followed later by the major illness—the major illness being characterized by neurological signs or paralysis.

At the time of the *minor* illness or the abortive form of polio as it is sometimes called, neurological signs are therefore not found. The signs and symptoms of the minor illness may be those of a cold or sore throat. Sometimes the *minor* and *major* illnesses occur more or less simultaneously, and these two phases are telescoped, as it were, one into the other. Occasionally, especially in children, the first manifestation of an attack of polio may be that of a paralysed limb, or some other paralysis.

Poliomyelitis may be either paralytic or non-paralytic. Both forms are non-paralytic at the onset. It is only the passage of time which will determine which type it is going to be. There is little doubt that in some cases certain factors have a bearing upon whether or not the non-paralytic may become paralytic. One of these factors is certainly that of unusual exertion undertaken at the time of onset of the major illness.

Sometimes a patient thinking he is suffering from a chill or 'flu may try to "work it off".

As an example of this I would like to quote the following case history of an undergraduate who was admitted on October 6, 1950. He had been to London on September 30. He travelled to Cambridge the following day, October 1. He thought he had a "chill". Complained of headache and shivering and was out of sorts generally. The following day he sat an examination paper both in the morning and in the afternoon.

On the next day, October 3, the headache was more severe and he vomited. On the 4th the headache persisted but in spite of this he went for a day's shooting. On his return to College that evening he felt very tired and went to bed early.

On the 5th he had an oral examination in anatomy which he attended. He complained of severe headache, stiffness in his back and he vomited. Later in the evening he went down to supper—managed to get back to his rooms only with the greatest of difficulty. The following day—October 6—he was admitted to hospital, when he was found to have fairly extensive paralysis involving both legs, abdominals, bladder, lower intercostals, left shoulder. While he was in hospital he developed a segmental collapse of his left base. He weathered the storm, however, and was eventually transferred to an orthopædic hospital. When I last heard of him he was able to get about with the aid of walking calipers and a bath chair.

In his article Ritchie Russell (1947, *Brit. med. J.*) drew attention to the effect of severe exercise during the pre-paralytic stage on the subsequent development of paralysis and more recently in the *Journal of the American Medical Association*, 1950, Horstmann, in an analysis of 411 patients, confirmed the earlier conclusions of Ritchie Russell. She concluded that the critical time in regard to activity is the first twenty-four to forty-eight hours of the start of the major illness, during which the symptoms are very often mild.

Another factor concerned would seem to be tonsillectomy performed during an epidemic of poliomyelitis.

In reference to this I would refer you to the case of a family reported by Krill and Toomey in the *Journal of the American Medical Association*, September 1941, in which 5 of 6 children of the same family had tonsillectomy performed on August 22. By September 7 all 5 had bulbar polio. Three died within two days. The parents and the child, aged 2½ who was not operated on, had no symptoms.

There is I am sure little need for me to mention to you that it is now accepted that there is a definite relationship between the anti-pertussis or combined A.P.T. and pertussis inoculations performed during an epidemic and the subsequent development of paralysis in the limb injected.

Symptoms

I have made an analysis of the symptoms of these cases tabulated according to their frequency, which was as follows:

(1) Vomiting and nausea	67	(8) Constipation	13
(2) Headache	66	(9) Paralysis only	13
(3) Pains in the limbs	48	(10) Retention	9
(4) Neck stiffness	41	(11) Vertigo	9
(5) Backache	33	(12) Diarrhœa	8
(6) Sore throat	15	(13) Dysphagia	6
(7) Abdominal pain	14		

The first 5 of this group are mainly symptoms of cerebral irritation and I think it is important to bear in mind that polio is primarily an affection of the central nervous system.

There is invariably fever which may range from 99° to 103° or even 104° F. depending upon the severity of the infection.

In the examination of patients one finds that if the case is *not* one of long-standing there are signs of meningeal irritation such as nuchal rigidity, spinal stiffness, positive Kernig. In children a useful means of testing neck stiffness is to raise the head and shoulders off the bed by placing one's hands under the shoulders. In the presence of any degree of neck stiffness the head becomes extended and falls backwards remaining in a hyperextended position. One does not find in polio the same extreme degree of head retraction that one associates with meningitis of bacterial origin, nor does one find that photophobia is so often a feature.

Superficial and deep reflexes must be examined. In an incipient paralysis these will be diminished—in a fully established paralysis they are absent.

It may be on the other hand, especially in the case of children, that the case has progressed beyond the stage of meningeal irritation. This may have been slight or entirely missed, and all that one finds clinically is a flaccid paralysis with absent reflexes.

Obviously one should test for paralysis in an orderly way, starting with the cranial nerves and working down to the feet.

In my experience the cranial nerves most frequently involved are the VIIth and Xth though none apart from the Ist is immune.

The signs and symptoms of cranial nerve paralysis may be difficulty in swallowing, nasal phonation, facial paralysis, paralysis of ocular muscles, or of the tongue or of the muscles of mastication.

It must be remembered that a pharyngeal paralysis may appear at first sight to be a respiratory embarrassment, the difficulty in breathing being due to the accumulation of mucus and saliva in the pharynx. In some cases also these secretions, which the patient is neither able to swallow, nor to spit up, gravitate towards the larynx causing some adductor laryngeal spasm, and further respiratory embarrassment. On the other hand a respiratory paralysis may appear to be a bulbar. The patient, owing to the respiratory paralysis, is not able to cough and consequently the sputum accumulates in the pharynx.

Having examined carefully for evidence of paralysis, see if the patient can sit up in bed unaided. It may be that he can only sit in the so-called tripod position. This means that he or she is only able to sit up with the support of the arms, usually placed some distance behind. If the patient can place the head between the knees there is usually no spinal or neck stiffness to worry about.

In the non-paralytic type of case the signs and symptoms are those of an ordinary meningitis. I will have more to say in regard to this later.

I would like now to describe a case which shows the difficulties which can arise in dealing with a case of *bulbar paralysis*.

This man, aged 29, was admitted on September 27, 1949, with a temperature of 100°, marked meningeal signs, nuchal rigidity, spinal stiffness, positive Kernig's, no evidence of any cranial nerve involvement at this stage, or of any muscular paralysis.

Superficial and deep reflexes normal and physical examination otherwise negative.

Lumbar puncture revealed clear colourless fluid, cells less than 3. Protein 75 mg.

On the 29th, thirty-six hours after admission, he had some difficulty in swallowing and developed a paralysis of his left facial nerve. That afternoon a feeding tube was passed. He had an uncomfortable night during which he removed the nasal feeding tube. Next morning his condition became much worse, and the symptoms of his pharnygeal paralysis were much more acute. He was having respiratory failure due to the accumulation of unswallowed secretions which continually interrupted the free flow of air necessary for respiratory interchange. He had some cyanosis, pulse 116, and was generally in a very distressed condition. He was laid flat in bed, the foot of which was raised.

All attempts to replace the feeding tube failed. An endotracheal tube was passed down the nasopharynx and continuous suction was started. As these unswallowed secretions in the pharynx were now being disposed of, the patient gained considerable comfort.

Further attempts at passing a feeding tube repeatedly failed. An intravenous drip was set up for two days and it was then decided, in consultation, that owing to the impossibility of forecasting the duration of the pharyngeal paralysis and the possible problem of feeding that a gastrostomy should be performed.

On October 3 a gastrostomy was performed under local anæsthesia. He made good progress, his pharyngeal tube being changed daily.

It was noticed on October 8 that he was able to swallow his secretions. Continuous suction was discontinued, he made further progress, and was gradually given semi-solid food, and his gastrostomy tube was removed on the 13th.

He made an uninterrupted recovery and was discharged on November 2 with only very slight evidence of a facial paralysis, from which he eventually made a complete recovery.

This case shows how serious complications may arise from what was a paralysis of relatively short duration, and the importance of trying to do something for the patient to tide him over this critical period and the ample reward for so doing.

I do not use continuous suction in all cases of pharyngeal paralysis. It depends on the degree. If the paralysis is moderate it may suffice to tilt the foot of the bed then fix on to the suction tubing an ordinary dental saliva ejector and from time to time the nurse or the patient uses this to remove excessive secretions.

Lumbar Puncture

I have tabulated the result of C.S.F. findings in both the paralytic and non-paralytic cases. In this series there were 81 paralytic cases and 19 non-paralytic.

Lumbar puncture was performed in 68 of these paralytic cases. Sugar and chloride content of the C.S.F. were found to be normal as is the case in poliomyelitis. 9 of these cases out of a total of 68 had a cell count of less than 5, and a protein content of less than 40, giving a percentage of slightly over 13 of the paralytic cases with a normal cell count and a normal protein content.

In regard to the 19 non-paralytic cases occurring in the series, 3 out of the 19, or a percentage of about 15, had a normal cell and protein content. This gives a total of 14% in the whole series with a normal cell count and a normal protein content.

I think this is something that should be borne in mind as the question often arises as to whether one can get an acute polio with a normal C.S.F.

Differential Diagnosis

It is not possible clinically to diagnose polio at the stage of the minor illness. There may be suspicious signs and symptoms such as a sore throat, headache, muscular pains, nausea. There is, however, no means of diagnosing this clinically as poliomyelitis even if it be such.

In the non-paralytic there are the signs and symptoms of meningeal irritation such as are found in

meningitis of bacterial origin including tuberculous meningitis. A meningism associated with a pneumonia must also be excluded. However, as I have said before, there is not the same degree of head retraction in polio as in meningitis of bacterial origin. Photophobia is also an uncommon finding in polio.

Lumbar puncture and examination of the C.S.F. will help to exclude such a bacterial meningitis. The history will help to exclude the encephalitis following measles, chickenpox and vaccinia. Mumps meningitis, whether or not the salivary glands have been swollen, must be thought of, especially if there is an epidemic of mumps in the community. I have seen 2 such cases. I believe also one can get a cranial nerve palsy associated with cerebral tetanus.

Other virus infections, such as lymphocytic choriomeningitis and coxsackie virus infections, are described as causing a similar meningitic syndrome though I have no personal experience of these conditions.

In regard to paralytic polio, a pharyngeal paralysis may at first sight simulate an acute throat infection such as a quinsy or a retropharyngeal abscess. This may perhaps seem strange but I have seen 4 cases of pharyngeal paralysis which prior to admission were thought to be acute throat infections.

In both conditions the patient may have difficulty with phonation. In the case of a quinsy this is due to pain and to swelling of the soft tissues. In the case of a bulbar paralysis, however, it may be due to laryngeal paralysis.

In the case of a pharyngeal paralysis it may be difficult to inspect the throat and pharynx because of the presence of the accumulated secretions. It can, however, be seen that there is no swelling of tonsillar tissue or of the surrounding structures. There is no œdema, no grossly infected tonsils, no exudate, no adenitis.

The dyspnœa of a respiratory paralysis may be thought to be due to a pneumonic condition. The patient may look very distressed and may be slightly cyanosed. On closer examination, however, it will be noticed that there is little or no movement of the chest wall.

A respiratory paralysis may occur as a primary paralysis of the respiratory muscles or it may be combined with a bulbar paralysis or be due to a disturbance of the respiratory centres. If both deltoids are paralysed there is nearly always a respiratory paralysis. *This is most important. In fact, all the serious respiratory paralyses I have seen had a bilateral abductor paralysis of shoulders.*

When the spinal cord is the main focus of virus infection we get flaccid paralysis of various muscular groups depending on the area or areas of the spinal cord involved. This paralysis is associated with absent reflexes and there are no sensory changes.

There are various local conditions which may simulate paralysis such as adenitis, osteomyelitis, synovitis, arthritis or a spinal disc lesion.

Another misleading condition in children may be the joint changes in rheumatic fever.

All these may lead to a limb being held immobile.

In these conditions, however, the tenderness will be more localized and the reflexes, apart possibly from a disc lesion, will be normal.

Infectious polyneuritis is a condition which may simulate polio most closely. Here the paralysis is usually symmetrical, associated with sensory changes and there is a relatively very high protein in the C.S.F. Recovery is more rapid than is the case in polio.

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Dr. F. O. MacCallum gave a résumé of recent advances in knowledge of the properties of the virus and techniques for laboratory diagnosis of poliomyelitis. He also showed tables of the number of cases notified and the attack rates for different age groups since 1944–45 derived from information supplied by the Epidemiological Section of the Ministry of Health. Attention was drawn to the fact that there were several hundred cases in infants under 1 year of age in the epidemic years of 1947, 1949 and 1950.

Dr. MacCallum also discussed the possible reasons for the high proportion of notifications of non-paralytic cases in 1951 when the total notification rate was only about ¼ to ⅓ of the recent epidemic years. He suggested that these might have been due to:

(1) Some other virus or viruses as yet unidentified.

(2) Different strains of poliomyelitis virus from those to which the patients may previously have been exposed and thus produced an attenuated attack or else an attack might have been attenuated by a previous minimal immunity of the same strain.

(3) Environmental conditions, such as the atmosphere, might have had some effect upon the virulence of the virus and its effect on the host.

[*February* 20, 1952]

DISCUSSION ON PROFIT AND LOSS IN PROPHYLAXIS

Dr. Lindsey W. Batten: That prevention is better than cure is a truism which can scarcely be gainsaid and, as a basic principle, prophylaxis must and does commend itself to all right-thinking doctors. Unquestionably we wish our patients to be well and are not diverted from the pursuit of their health by any "vested interest in disease". Yet not all of us feel our hearts leap up when pressed to give or do something intended to protect our patients against an ailment they have not yet got.

We may feel that the time, money and effort spent in prophylactic procedures might be more fruitfully employed in providing means and incentives to healthy living or even in the use and enjoyment of existing health, and we may also be conscious of the fact that prevention, like everything else, has its price. For each act of prophylaxis there is an account to be cast.

To cast this account accurately is often impossible. Unknown quantities may exist on both sides of it. But far too often only the credit side is mentioned or even perceived. Debits go unrecognized. I wish to propound examples of familiar prophylactic procedures in some of which I believe this balance to be doubtful or adverse.

Prophylaxis can be general or individual. Even our piped water supplies and methods of sewage disposal have items on the debit side of their accounts that we rarely consider—but general measures do not specially concern the practitioner and I shall here limit myself to things done, given or advised by doctors to protect individuals against some malady or loss of health not yet suffered. I propose further to take my examples roughly in the order in which they may occur in an individual life.

Antenatal

Before birth the infant may find himself turned from a breech to a vertex—all gain and no loss; but should his mother have rubella before the twelfth week of his life he may find his existence terminated for fear he should be born with a congenital defect and here the state of the balance may seem to be in doubt.

Neonatal

Three prophylactic assaults confront the newborn babe—two common, one becoming rare: vaccination, circumcision, and division of the *frenum linguæ*. Vaccination is clearly prophylactic. The other two might claim to be treatment, for phimosis and tongue-tie respectively. But it must be rare for either operation to be performed for the relief of symptoms actually arising from the alleged abnormality. Babies are circumcised, ritual apart, to avoid "trouble", not very clearly specified, later on. Their tongue-strings have been divided lest they be "tongue-tied" when they come to speak. It is assumed that the short frænum, inserted near the tongue's tip, will retain this character, that the foreskin will remain adherent and narrow. As for the frenum, it is generally acknowledged that it will lengthen and that the forepart of the tongue, developing late, will come to project well beyond its insertion, so that a recent textbook can justly describe tongue-tie as "this myth of hoary antiquity". There is, in fact, widespread agreement that division of the frenum is, or was, a piece of surgical prophylaxis showing a loss.

Prophylactic circumcision cannot be so summarily condemned but its case is not dissimilar. The foreskin, like the tongue, is commonly but half developed at birth. According to Gairdner (1949), it will, in the vast majority of cases, if left completely alone, free itself from the glans and become retractable in babyhood or infancy. Persistent phimosis is rare and the operation is almost never surgically necessary. The slight hygienic advantages of having no foreskin should be weighed against surgical trauma, sometimes without anæsthetic, at an age of adjustment to a new environment, and the known possible misadventures at or after an operation apparently responsible for some sixteen infant deaths a year. Occasionally the circumcised have to regret the loss of a useful reserve of elastic skin for autogenous grafts.

Infant vaccination has abundantly justified itself in the past and is undoubtedly a part of good citizenship. It also reduces the discomforts of any later vaccinia; but the practitioner who, in some forty years of medical experience, has not once seen major smallpox, may perhaps be forgiven if he vaccinates the hitherto unblemished baby without enthusiasm.

Postnatal

There is no doubt about the credit balance in diphtheria inoculation. The debit side, if the injections are given between six and nine months, is almost a blank. Whooping-cough vaccine, if effective in combination with diphtheria prophylactic, as it is now reported to be, or if at least it can be usefully given in the same early months, has also a good credit balance; but immunizing injections given at a time of life when consciousness is awake but reason still slumbers have debits to be reckoned with. It is undesirable and may be serious to inoculate into a child a distrust and dislike of his doctor—perhaps even of the whole medical profession, and three needle stabs in the second or third year of life are well calculated to have this effect. Some little "toughs" take no notice or readily forgive, but to lose the trust and affection of a sensitive child-patient is a considerable price to pay for damping

down the whooping-cough. What is to be done if our eager, industrious scientists devise more and more immunogens, making infancy one long series of needle pricks? Can we perhaps contrive that all injections between years 1 and 6 be given at public clinics, hoping thereby to provoke in tender minds a wholesome distrust of these institutions with a countervailing warm regard for the family doctor?

Inoculations over, the child may well escape active preventive measures until he goes to school. Once there he is almost sure to find himself one day suddenly "in quarantine". The child he had tea with last week developed mumps, rubella or chicken-pox a day or so later. In consequence he may not go to school, his friends regard him afar off, his social activities and those of his family are seriously curtailed and his mother is in telephone consultation with his doctor. Blame-fixing, fault-finding and anxiety are in the air about him. Individually we may think little of quarantine for contacts but collectively we are responsible for it. Presumably it ranks as prophylaxis, for if not, then what is it? But whom are we trying to protect and from what?

The only principle deducible from our behaviour seems to be that if we cannot prevent an illness it is always our duty to postpone it. We act as if it were better to have what may reasonably be called the "inevitable fevers" in adolescence or adult life than in childhood.

This really will not do. Adult and adolescent males get orchitis from mumps; infant males do not. A woman with rubella may be carrying a susceptible embryo; not so an infant girl. Severe chicken-pox, when I have seen it, has been in adults and whereas any of these ailments may cost their adolescent or adult victim a scholarship or a job they cannot do this to the infant.

On these and other counts it is, without qualification, best to have these fevers young and this often means "have it now". No doubt measles is best postponed until after 3 and scarlet fever—if it counts as an entity—altogether if possible; but gamma globulin and penicillin, judiciously employed, are better weapons for combating these two diseases than the clumsy device of quarantine for contacts. How futile and how wasteful this contact-quarantine is has been conclusively demonstrated by the experience of Rugby school. I am sure it is significant that this twice-published experience is still not widely known. Rugby abolished quarantine for contacts about 1925, in the time of R. A. Simey. Thenceforward all contacts returned after the holidays. In 1943 R. E. Smith reviewed in the *Lancet* the results of the previous ten years. In this time 128 known contacts had returned to school, some artificially or naturally immunized to the disease in question, others unprotected. 2 only became cases, and even these not sources, of the disease. The days of quarantine, calculated at textbook rates, for *all* these contacts numbered 2,615; for the unprotected boys alone, 1,356. Some quarantine days would have fallen in the holidays but most in the term, so the profit in boy-school-days was well over 1,000. Further experience, recorded in 1947, confirmed these results. By then the quarantine days for the unprotected amounted to 2,123. By then, also, there had been 73 outbreaks of infectious disease in the sixteen years of observation, *not one* traceable to a known contact. 3 only of 203 known contacts developed their disease. Surely the publication of this resounding success should have finished the "health certificate"—at least for boarding schools. But it has not; boarding schools still demand it. The L.C.C. has steadily relaxed its precautions for day schools but private day schools often have stringent rules and to hand on chicken-pox can be an offence mortal to family friendship, though a gift of influenza or a virulent cold may be made without blame.

I believe that the complete abandonment of quarantine for the lesser infectious fevers, except possibly in day-nurseries and nursery-schools, would pay a handsome dividend in terms of child-school-days, happy social occasions, good neighbourliness and a cheerful and courageous outlook on life.

Next may come prophylactic tonsillectomy.

Tonsils are not, like vermiform appendices, removed *in flagrante delicto*, so every tonsillectomy could claim to be prophylactic. But there is a difference between removing tonsils guilty of repeated tonsillitis or quinsy, or constantly and visibly in a state of septic infection, with enlarged lymph glands and impaired general health, and the removal of enlarged but quiet tonsils to *prevent* colds, rheumatism, otitis media, or some undefined lapse from health which is to occur if they are left, or tonsillectomy as a mere adjunct to a necessary removal of adenoids.

That many hecatombs of tonsils have been offered on the altar of prophylaxis we all know; to judge by waiting lists the goddess and her ministrants are still not satisfied; yet they would be hard put to it to find justifying evidence. Alison Glover in 1932, after an investigation which included a school population of nearly 14,000, reported as follows: "While the incidence of recurrent sore throats is perhaps slightly diminished that of frequent colds is unaltered or perhaps slightly increased. The incidence of otitis and mastoid disease is the same or perhaps slightly increased upon the tonsillectomized, while their liability to bronchitis and pneumonia is also probably slightly increased." I think it would be fair to say that Glover found no justification for prophylactic tonsillectomy at all. In a later paper (1948) he records that "at least 85 deaths of children under 15 occur on an average each year from tonsillectomy and in all probability this is a very conservative estimate". There are many more items to be set down on the debit side of the account and had not poliomyelitis been in abeyance at the time of Glover's report the case for restraint would have been stronger still. Yet

it was, one would have thought, strong enough and it is distressing to find Dr. Glover having to repeat the substance of it to a county Pædiatric Society fifteen years later.

For myself, I have seen and noted the excellent results of well-judged and well-performed *therapeutic* tonsillectomy, the frequent failure of *prophylactic* tonsillectomy to deliver the expected goods, and many of the misadventures that can follow this operation. I notice that many adults know no reason for having lost their tonsils and are surprised that they should be expected to know one. Outside my practice I had for nearly twenty years the experience of inspecting London school children, and the school doctor has outstanding opportunity for observing the natural history of the tonsil. As the years passed my respect for the *vis medicatrix Naturæ* grew and grew. So many tonsils, not only large, but with the stigmata of infection, put my judgment to shame by becoming, so far as I could see, perfectly normal in two or three years with no treatment save the passage of time. I suggest that here again we have not cast the account. Dazzled by the glamour of prophylaxis we fail to discern the facts. How else explain 200,000 annual tonsillectomies in this country about 1938?

Next comes an example of the most innocent-seeming yet most questionable of all the prophylaxides—the attempt to forestall disease by diligent search for its first flickerings, by repeated inspection and by restraints. Systematic endeavours to control acute rheumatism in childhood and to prevent its sequelæ by these methods have been made in London in the last twenty-five years. I have come into contact with the workings, effects and side-effects of these endeavours in schools, in two children's hospitals and in private practice, and I feel most doubtful whether the profits and losses have been justly assessed. Rheumatic carditis is a disease which can take or cripple a young life and which admittedly smoulders with outbursts. It can work openly or, it seems, insidiously. We have all seen the recruit or schoolboy with every classical sign of an established mitral stenosis, often symptomless, and no history to show how he came by it. On our present knowledge we are bound to assume a past rheumatic carditis and, pressing hard with leading questions, we perhaps squeeze out a reluctant history of limb pains long ago. We all admit that rest in bed is good for carditis and may believe, though we could scarcely prove, that timely and continued rest may stay the progress of the disease and save a valve. It has been argued that had not such a boy's symptoms been disregarded his heart might well have been saved.

It is, I think, on this basis of argument that the scheme for prevention has been erected. Parents, teachers, doctors, even children themselves, are urged to note and take action on the earliest symptoms chorea is bracketed with rheumatism and all too often in the past fidgets has been equated with chorea and limb pains with joint pains or arthritis. A child who fidgets, twitches or has transient pain in thigh or calf is submitted to searching and repeated medical examination; an evening temperature of 99° or a systolic præcordial bruit has been held to justify such diagnoses as "potential rheumatism" or "pre-rheumatic state". A child in such a category may find himself forbidden to play games, kept out of school, put in a hospital bed for three months or merely re-examined every four weeks. At last he may be discharged but it cannot have escaped him that the integrity of his heart has been in doubt.

In future this may happen less often. A more tolerant view is taken of limb pains and I trust it is widely recognized that fidgets and tics are not precursors of rheumatic chorea. A normal sedimentation rate may acquit a "thermolabile" child of the charge of rheumatic fever. But very many children have been subjected to prolonged observation and curtailment of activities in the name of preventive medicine in the last two or three decades. Has it paid? No one can know. It would be hard to prove that a single heart had been saved by resting or restraining the "borderline case", but assuming that many have, there is still the cost to be set down. It consists first of the lost school-days and lost child-play-hours, which must total thousands over the years, and next of something invisible, impalpable but real and known to us all in our consulting rooms—loss of confidence in the integrity or reliability of that vital organ, the heart. It is said that to keep these children under periodic observation "can do no harm". To a very phlegmatic, extrovert child, with parents of the same type it may do none. To others it may do lifelong harm. Few, it sometimes seems, not in general practice understand how common, disabling and intractable is "nosophobia" and how much is at least partly "iatrogenous"-doctor-engendered fear of disease. It is not only that, to quote Langdon Brown, "every patient who comes to see us is afraid"; it is that many are suffering from fear, in one of its innumerable manifestations, and from nothing else. Cardiac neurosis is, in my experience, commoner than valvular lesions; no "compensation" occurs; treatment is long and difficult. Is not our one duty to these children to make a diagnosis of "carditis" or "no carditis" in the shortest possible time—surely a fortnight in hospital would almost always suffice—to rely on the diagnosis made and act accordingly?

If the conclusion be "no carditis" I suggest the child should be discharged not to periodic observation but *altogether* unless definite symptoms occur. Surely the risk of promoting a neurosis by observation far outweighs the risk of failing to prevent a preventable cardiac lesion.

Adolescence

So much for the common prophylactic proceedings of childhood, considered in some detail; others, affecting those of riper years, must be dealt with more generally. First, routine inspections. I believe, if all were known, a credit balance in childhood and youth would be found to change slowly

to a debit after middle life. In youth such things as squints, defective sight in one eye or both, crowded or decaying teeth, postural or skeletal faults—things nearly symptomless, yet of moment and remediable, may be found and set right and the mere fact of being stripped and examined has a salutary influence. Against this must be set the baseless alarms, the anatomical accidents erected into diseases, which every family doctor knows too well and, of course, part of that indecent mound of excised tonsils lies at the door of school inspections. It would be a bigger part but for the sturdy "sales resistance" of working-class parents.

Adulthood

I surmise that the examination of recruits and young soldiers has a substantial credit balance, though the rejection by recruiting boards of young men with perfectly compensated valvular lesions or a history of duodenal ulcer must have spoiled some good lives; but as we get older the chance of a profitable discovery sinks, the chance of a regrettable revelation grows and grows. We stiffen and get set in our ways; we bear honourable or dishonourable scars; inevitable degenerations begin. We are sure to have skeletons in our cupboards best left inside. It is a doubtful policy to invite medical examination, in the absence of symptoms, after middle life.

I know American life insurance societies believe that routine examinations prolong life. They may. But there is the world of difference between prolonging life and promoting health. Insurance companies are concerned solely with longevity, "as though to breathe were life", not with health and happiness. I feel grave misgivings when any patient over 45 makes advances to a life insurance company.

Next come *ad hoc* procedures—mass X-raying of chests, antenatal examinations, periodic overhauls to detect symptomless cancer. Of mass X-ray I have little personal experience; I have an impression that the balance is favourable. Everyone, I think, agrees that antenatal examinations yield a profit, though less substantial than was once hoped. Probably few general practitioners favour cancer-divining operations. The trouble is that a really symptomless cancer almost always defies detection by clinical means until it is inoperable; it is not practicable to employ all the diagnostic aids every six months, so, on any given occasion, we can only say "I find no evidence"; and the subject's moral fibre must be tough indeed if he or she is not to feel some dread of the next examination two or three months before it falls due. Alternatively he may overrate the last reassurance and neglect warning signs. I believe we should confine ourselves to urging early attention to likely symptoms and to teaching our patients what likely symptoms are. They still do not know.

There remains a curious group of things to be swallowed, injected or applied to promote general or specific immunity—curious because its members vary so extremely in value. It contains such justly famous winners as vitamin D for rickets and T.A.B. vaccine for the enteric fevers, cheek by jowl with such "also rans" as anti-catarrh vaccines to banish colds and capsules of mixed vitamins competing with ultraviolet light as universal protectors and elixirs of life.

Doubtless the good done by the "winners" in this group, even disregarding victories over tropical diseases, far outweighs any bodily harm done by the rest. But losers and "also rans" are deplorably hard to get off the course. Carefully controlled field research, proving the emptiness of their claims, is fully reported in the journals, but it never reaches the lay press and on they go until they die of exhaustion. The cost here is in the misuse of skilled labour, machines and raw material and in the slur cast on the scientific basis of medical practice. Here, then, are some examples, chosen without bias, which at least illustrate the thesis that individual medical prophylaxis is, like marriage, "not to be enterprised or taken in hand unadvisedly, lightly or wantonly" and that, in fact, it has its dangers.

What is the actual balance, in terms of mind-and-body-health, of all these attempts to avert anticipated disease cannot be certainly known but I am certain that too often their probable—or even certain—cost is not appreciated. In accordance with the spirit of our age we are strongly and repeatedly urged to assume prophylactic and health-giving powers greater than we possess. I believe we do ourselves and our fellow citizens an ill service by accepting too readily this conception of our functions.

Our primary task is still our traditional one—diagnosis, prognosis, treatment, the care and comforting of the sick. When we can safely and surely prevent we must, of course, do so, but we should first do all we can to calculate the cost.

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Sir Wilson Jameson (late C.M.O., Ministry of Health) pointed out that individual prophylaxis was never so dramatic as were the results of some forms of modern therapy. All the same, the effect upon the community of mass individual prophylaxis was at times remarkable.

Though Dr. Batten had dealt very fairly with the pros and cons of tonsillectomy, he had made no adequate reference to its social implications. Dr. Glover, the authority on the subject, had shown that, in pre-war days, some 50% of girls and 58% of boys entering a number of the larger boarding schools in this country had been subjected to the operation as compared with a figure of about 20% for elementary school children during the whole of their period of school attendance. In short, Dr. Glover said "for a boy to be born with a silver spoon in his mouth seems to be one of the conditions that lead to tonsillectomy".

As regards specific methods of prophylaxis the results in diphtheria and in smallpox spoke for themselves and it seemed as though a potent vaccine against whooping cough was becoming available. Family doctors could do much to influence parents in favour of these prophylactic measures and it was of interest that between 1947 and 1951 the percentage of diphtheria immunizations performed by general practitioners had risen from 28 to 47.

Some of the procedures labelled by Dr. Batten as prophylactic were not infrequently "safety play" on the part of the doctor, e.g. restriction of activities in the case of doubtful cardiac lesions. The retention of out-dated quarantine rules seemed to be due to the same sort of attitude on the part of the school managers or other responsible persons.

If it was true that a certain amount of "nosophobia" was due to doctors doing or saying too much to patients, a good deal of anxiety in patients and in their relatives was caused by too secretive an attitude on the part of doctors. The public were becoming much better informed and the medical profession must be prepared to satisfy their legitimate inquiries. With most people, to know more was to fear less. Education of the public in the early signs and symptoms of cancer was to be commended if no more was promised than could be fulfilled. There was a good deal of U.S.A. experience in this field and it was stated that in Massachusetts, between 1935 and 1948, the average delay, for all types of cancer, between the date signs were first noticed and the date of the first visit to the doctor declined from 6·2 to 3·9 months—a period characterized by an active educational campaign supported largely by the doctors throughout the State. The practice followed by a family doctor in the north of England was mentioned—some 50% of his female patients between the ages of 40 and 70 had accepted appointments for a clinical examination, and 330 had so far been examined. One breast carcinoma and a number of gynæcological conditions had been detected. Some patients were being kept under observation. The same doctor did what he could to provide his patients with simple leaflets of a health educational character in the hope that this might reduce to some extent the demand for "the bottle of medicine". He was finding the work rather uphill and disappointing.

Medicine, both curative and preventive, was having the most productive period in its history. General practitioners were well aware of the results of the newer forms of therapy. In the field of prevention the gains were no less outstanding. They were attributable in the main to our better means of control of infection and to improved nutrition. Even tuberculosis was now on the retreat. From 1948 to 1949 mortality from this disease declined by no less than 10% and from 1950 to 1951 the decline seemed likely to be in the order of 14%. It had been stated that only one death from diphtheria occurred in the L.C.C. area during 1951. The children's wards in many of our general hospitals were far from full. Indeed, the pattern of hospital in-patients was altering under our eyes. All this had not come about by itself—all sorts of people and all sorts of agencies had played a part, not least the family doctor. In spite of the rather pessimistic note struck by Dr. Batten at the end of his paper, it was clear that the family doctor had been given more and better opportunities to share in the practice of preventive medicine and all the evidence went to show he was taking advantage of the opportunities offered.

Section of Proctology

President—CLIVE BUTLER, F.R.C.S.

[*January* 30, 1952]

The following Cases and Specimens were shown:

Mr. RUPERT CORBETT.

(1) **Colon and Terminal Ileum showing Regional Ileitis and Regional Colitis with Stricture Formation** from a woman of 47 with intestinal obstruction. Following a subtotal colectomy rectal control was re-established. (2) **The Fate of a Dragstedt's Ileostomy.** Four months after formation, the ileostomy completely disappeared into the abdomen. Function was maintained and on re-fashioning it was found that owing to a stenosis at the opening, the ileum was drawn in with separation of the graft and, finally, the tube of the graft was also in the abdominal wall.

Mr. A. DICKSON WRIGHT.

(1) **Plastic Carcinoma of Cæcum Complicating Chronic Colitis** of twelve years' standing (man of 26) complicated by nephrosis and anasarca. The colon though greatly fibrosed was not polyposed or ulcerated, although mucosa was completely changed in character and appearance. (2) **Plastic Carcinoma of Rectum** from man of 84, resembled plastic linitis of the stomach and mucosa. Membrane was normal and unulcerated. Recovered well after abdomino-perineal resection. (3) **Ileostomy and Subtotal Colectomy for Ulcerative Colitis.**—The patient (female of 23), after nine months' illness, had lost half her weight at time of operation (4 st. 7 lb.). A total colectomy, with spout ileostomy carried out in one operation in spite of emaciation, had a remarkable effect and she now weighs after two months, 7 st. 10 lb.

Mr. LIONEL E. C. NORBURY.

Perforated Diverticulum of the Descending Colon Simulating a Carcinoma in a Man Aged 44 Years.

Mr. W. B. GABRIEL.

(1) **Rectum Showing a Carcinoma and Lymphosarcoma at the Same Level.** (2) **Squamous-cell Carcinoma of the Rectal Ampulla.**

Mr. H. E. LOCKHART-MUMMERY.

Squamous-cell Carcinoma of Recto-Vaginal Septum.

Dr. JAMES EARLE.

(1) **Malignant Growth of Rectum of Indeterminate Origin.** (2) **Unusual Spread of Mucoid Carcinoma of Sigmoid Colon.**

Mr. STANLEY AYLETT.

Anus, Rectum and Lower Colon from a Patient Suffering from Non-specific Ulceration of These Regions.

[*February* 27, 1952]

The Use of Bradosol Solution (1 in 2,000) as a Wound Dressing in Rectal Surgery

By W. B. GABRIEL, M.S., F.R.C.S.

BRADOSOL is a new antiseptic, a quaternary ammonium compound, produced by the Ciba Research Laboratories. It is described as a cationic bactericide and detergent, readily soluble in water and giving a neutral solution which lathers copiously. Bradosol is stated to be effective in very low concentrations and to have a Rideal-Walker coefficient of 450. Bacteriological tests have shown that it has a high degree of activity against *Staphylococcus aureus* and *Streptococcus pyogenes*, and that it is also active against Gram-negative organisms.

In 1950 I thought it might prove valuable in dilute solution as a primary dressing for rectal wounds and, by virtue of its soapy characteristics, would be an efficient and kindly solution for the patient. Having in mind that dreaded third-day dressing which so often is tightly wedged into the anal canal, very adherent and only capable of being shifted little by little, with great care, under a stream of several pints of peroxide lotion, or else with the aid of intravenous Pentothal given in the patient's bed (a practice which I believe is not altogether without risk), I hoped that Bradosol might be the means of introducing an improvement in our routine. I accordingly agreed with the makers to give Bradosol an extended trial in rectal cases and I can say at once that my hopes have been realized.

This report covers a consecutive series of 310 minor operation cases under my care at St. Mark's Hospital from August 1950 to February 1952, inclusive. These were all in-patient cases in which open wounds were left at the anus following operations for cure of hæmorrhoids, fissures and fistulæ (either alone or in various combinations), partial prolapse, fibrous polypi, anal skin tags and abscesses. In all of these cases one or more flat open wounds were left at the anus. In the case of the fissure and fistula wounds a surface coagulant in the form of 20% tannic acid in flavine was dabbed on for a few seconds at the conclusion of the operation prior to application of the final dressing. This dressing, in all the cases now reported on, has been 1 in 2,000 aqueous Bradosol applied as usual on flat folded gauze with a corner of the moistened dressing tucked into the anal canal.

Post-operatively for the next two days the original gauze dressing has been damped each morning and evening with the Bradosol solution, and on the third morning the routine olive oil and gruel enema has been given.

This brings me to the crux of this report. Previously the third-day dressing has been a painful and sometimes a distressing session, but recently I attended at St. Mark's Hospital one morning with the intention of seeing what exactly happened to 6 patients operated upon three days previously—4 of these were fissures (surely the test of any rectal routine), and 2 were straight hæmorrhoids. In all of these cases I found that the original gauze dressing had been passed spontaneously, definitely without any traction by the nurse or sister, in 5 cases after the enema and in 1 case before it. The third-day dressing then merely consisted in irrigating the wound with peroxide lotion and tucking in a fresh dressing. I feel quite sure that these patients had a very comfortable passage with their third-day dressing compared with the average. Since making this observation a careful note has been kept regarding the third-day dressing in a total of 43 rectal cases, and it has been found that in 37 (or 86%) the original dressing came out spontaneously; in only 6 cases was it slightly adherent so that some traction under a stream of lotion was required.

Subsequently the dressings have been carried out on routine lines by irrigation morning and evening with dilute peroxide lotion, and a fresh gauze dressing moistened with 1 in 2,000 Bradosol has been laid on the wounds.

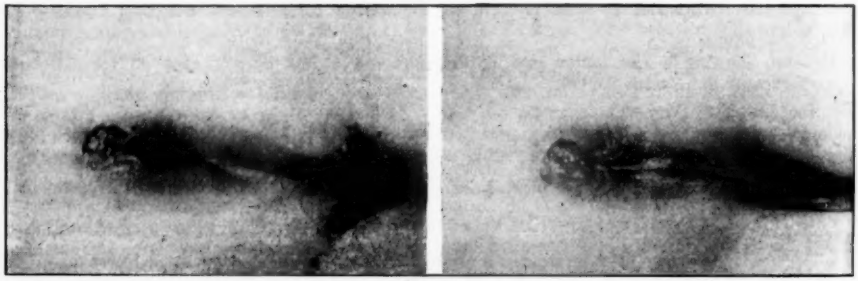

FIG. 1

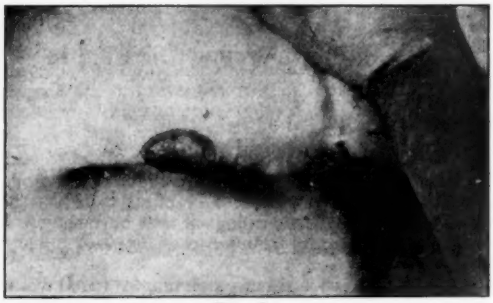

FIG. 2.

FIG. 1. — Two fissure wounds on the fourth post-operative day, showing the clean wound surfaces, the clear-cut edges and absence of skin irritation after dressing with Bradosol lotion.

FIG. 2.—Fistula wound on sixth post-operative day showing remarkably clean surface and margins on Bradosol dressings.

Later results.—I have no hesitation in saying that the state of these anal wounds, on and after the third day, has been superb, as will be seen in Figs. 1 and 2. There has been no skin irritation, and on the third day the skin margins have been clean cut, without the least sign of inflammation, and the surfaces of the wounds, particularly in the fissure cases, have been amazingly bright, clean and relatively free from evidence of the tannic acid coagulation.

The subsequent course has been equally satisfactory and when inspected on the sixth post-operative day the wounds have been flat, clean, and in my estimate several days in advance of the average. In the early days of this series, while the Bradosol solution was still under trial, the dressings were usually changed to lotio rubra on the sixth or seventh post-operative day, but now the wounds seem to go so well if kept exclusively on Bradosol that I seldom see any indication for making a change. Many fissure cases have been discharged on the sixteenth post-operative day and have only needed one subsequent visit to the Out-Patient Clinic.

The hæmorrhoid cases constitute slightly over half of the total number treated with Bradosol (162 out of 310 cases) and have done extremely well; the anal wounds have kept remarkably clean and healing has now become very even and rapid, without any evidence of that initial period of local sepsis and discharge which used to be quite common after hæmorrhoidectomy, in old subjects especially.

Bradosol solution has been particularly effective in cleaning up big fistula wounds. In 2 recent cases of infected pilonidal sinuses with secondary discharging openings which were excised and left open, the wounds were dressed with Bradosol on and after the third day when the original zinc peroxide gauze was removed. Although these wounds were rather large and deep at first, they are healing very quickly and present ideally bright red clean surfaces. The patients are very comfortable and have had very little pain.

Conclusion.—I can without hesitation recommend 1 in 2,000 Bradosol as a primary dressing for rectal wounds and its soapy characteristics provide many advantages: it is a good antiseptic and seems to inaugurate rapid wound healing by granulation. In the series of 310 cases now reported I know of no instance in which the skin has become inflamed or irritated, or in which the use of Bradosol has had to be discontinued or changed for this reason.

As a postscript to the above I might add that Bradosol is equally satisfactory for a perineal dressing after excision of the rectum and it is also being used for the subsequent dressings after that common out-patient operation of excision of an anal hæmatoma.

Alcohol Injections in the Treatment of Pruritus Ani

By J. H. Lees Ferguson, M.B.E., M.A., M.B., B.Chir., F.R.C.S.

It is generally recognized that the majority of patients suffering from pruritus ani have a local pathological condition or infection causing the irritation, which is susceptible to treatment (Gabriel, 1949).

In this short paper a residual number of 28 cases of essential, idiopathic, intractable or chronic pruritus ani are presented, in which no direct cause was discoverable, or in whom symptoms persisted after any probable causes were removed, and who eventually came to alcohol injection (Table I).

Table I.—Intractable Pruritus Ani
St. Mark's and Middlesex Hospitals. 1947–1951

Total cases reviewed	31
Too recent for follow-up	3
Total analysed	28
Ages: 24–70, majority of middle age	
Sex: Men 10, women 18	
Duration of Symptoms 1–22 years	
Average 7·7 years	

Table II.—Treatment of Associated Rectal Conditions

Hæmorrhoids injected	9
Hæmorrhoids excised	3
Fissure excised	2
Mucosal prolapse ligated	1

The multitudinous varieties of treatment advocated for this disease are sufficient proof of its unresponsiveness.

All of these patients received, for long periods, the basic general treatment accorded to all cases of pruritus, consisting of instructions as to careful hygiene, the application of simple preparations such as lot. mag. carbol. and prohibitions regarding diet and raiment. This resulted in slight improvement in individuals, but in no cures.

Local rectal conditions were treated in 15 patients (Table II).

No improvement resulted save in the patient with mucosal prolapse.

Other treatments of the pruritus are given in Table III.

Table III.—Direct Treatment of Pruritus

Hygiene and lotions (lot. mag. carbol.,* antimycotics, &c.)	28 patients
Ball's operation	3
Deep X-ray therapy	6 (10 courses)
Antihistamines	11
Psychotherapy	6
Proctocaine	7

* It consists of Phenol gr. xv; Zinc oxide gr. xxx; Prep. calamine gr. xv; Glycerine ℥ xxx; Rose water ℥ lx; Mixt. of mag. hyd. to 1 ʒ.

In a small experience Ball's operation and Krause's modification of it have not been successful in the treatment of any example of intractable pruritus. The benefits of X-ray therapy are outweighed by its dangers and disadvantages. Antihistamine treatment, so successful in America, has not given comparable results in our hands. Psychotherapy has been advanced as the logical treatment of a condition asserted to be predominantly psychogenic, but a psychiatric cure has yet to be seen in this condition.

Local injection treatment.—Of the oil base local anæsthetics, Proctocaine, A.B.A., and Benacol injections, as initiated and practised by Gabriel (1930), Naunton Morgan (1935), and Yeomans, Gorsch and Mathesheimer (1927) have all given periods of remission and cure in cases previously reported. Proctocaine injections in this particularly resistant group under consideration gave remissions of up to one month in 5 patients. One patient was made worse, and one had a remission of nine months.

Injections of alcohol.—All cases in this series received injections of 40% alcohol following the technique described by Buie (1931), except that more than one skin puncture was used in most of the operations.

The patients were admitted to hospital for periods of one to three days. They were shaved, bathed, and the injections were performed under general anæsthesia with full aseptic precautions.

The technique of injection consists in inserting a fine 12 cm. needle to full length immediately under the skin, parallel with its surface, and in the peri-anal space. A Labat syringe is attached, and alcohol slowly injected as the needle is withdrawn, the subcutaneous tissues being moderately distended in the course of the injection. The whole pruritic area, with special attention to the anal verge, is infiltrated in this way; the amount of 40% alcohol necessary varying between 20 and 50 c.c.

The placing of the needle is important, since intradermal injection will produce sloughs, and deep pooling of the solution is prone to cause cellulitis in the ischiorectal space.

The patient usually returns home on the day after injection, being instructed to continue the local and hygienic measures.

The results of injection are shown in Table IV.

The figures do not compare with Buie's (1931) 84% cure.

No analysis of reasons for relapse has been attempted, but it would seem that recurrence is far more common in patients in the lower income groups than those met in private practice, and may be related to differences in hygiene.

Furthermore, patients whose irritation was stated to be intense, but in whom the skin changes were minimal, only showed slight improvement. The treatment is by no means devoid of complications, shown in Table V.

TABLE IV.—RESULTS OF ALCOHOL INJECTION

28 Cases, 25 Followed-up

	Patients	%
Cure	10	40
Remission followed by recurrence (average period of remission 12·1 months)	5	20
Greatly improved, some irritation	5	20
Slightly improved, irritation still troublesome	5	20
Not seen again, apparently cured	3	

Number of injections: One —20 cases
Two — 7 „
Three— 1 „

Average amount of 40% alcohol used: 28 c.c.

TABLE V.—COMPLICATIONS

Superficial sloughing of skin	5
Cellulitis	3
Peri-anal abscess	3
Low level fistula	1
Transient subcutaneous sphincter weakness	2

Fortunately the complications are not such a drawback as they would seem. Sloughing of the skin can be avoided with care, and is of course painless. Cellulitis and abscess formation after alcohol are particularly prone to occur in female patients in whom the labiæ are involved by pruritus and have therefore to be infiltrated.

These inflammations do not pursue the same course as spontaneous pyogenic infections, presumably because the infection is of a low grade, and the contents of any localization are almost sterile. Early detection, and drainage by a simple stab incision, is sufficient to bring resolution and healing in a few days without hospital treatment. It is advisable, however, to warn the patient of the possibilities before treatment, and to see them within a week of their discharge.

It would be wrong to draw any far-reaching conclusions concerning this form of treatment from such a small number of cases, except to say that alcohol injection would seem to give a fair chance of cure or improvement in pruritus ani resistant to other methods.

The disadvantages of the treatment are first, that it is time-consuming and needs an hospital bed. We are hoping to overcome this objection by infiltration in the Out-Patient Department with 20 c.c. of 1% xylocaine and adrenaline, allowing this to diffuse for one hour, and following this local anæsthesia by the alcohol. So far, the injections have been painless and the results promising.

The second objection to this type of injection follows from the complications which may occur. The patients who have suffered them state that they consider the discomfort negligible compared with their previous itch.

Thirdly, and finally, it is unfortunate that cure cannot be guaranteed. This, however, is a state of affairs not unknown in other branches of the art of surgery.

May I thank all the members of the surgical staff of St. Mark's Hospital for their kindness in allowing me to include their cases, and in particular Mr. O. V. Lloyd-Davies for cases from his own practice and from the Middlesex Hospital.

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Some Aspects of Fluid and Electrolyte Problems in the Post-operative Period

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Middlesex Hospital, London

IT cannot be denied that in many of its aspects the problem of fluid and electrolyte balance in surgical cases is extremely complex, but at the same time it is true that modern ideas are leading to increasing simplicity in the practical management of such problems. This paper is only concerned with some aspects of the problems of the normal requirements of water and salt, and the replacement of observed losses.

The normal water intake of a patient is most easily considered by working backwards from the losses, which normally are by two routes, namely extra-renal and renal. The extra-renal loss consists of water lost from the skin by vaporization and sweating, from the lungs in saturation of the inspired air, and in fæces (normally less than 100 c.c. per day). Depending mainly on environmental temperature and on body temperature and activity, the extra-renal loss is enormously variable, lying between 750–1,500 c.c. per day: it consists of water containing only minimal electrolytes, is inevitable and has first call on the water available to the body. It is only after this extra-renal loss has been met that the remaining water is available for excretion by the kidneys. But with the water thus made available to them the kidneys have also to excrete at least 35 grammes of nitrogenous waste products per day and also any excess electrolytes. The amount of water required by the kidneys for this purpose depends essentially on their concentrating power. Normal kidneys capable of concentrating urine to a specific gravity of 1032 can excrete 35 grammes of waste products in about 500 c.c. of urine, but if their concentrating power is diminished by any process the minimum obligatory volume necessary to prevent nitrogen retention is markedly raised. It is generally agreed that 1,500 c.c. is the optimum daily urine output, giving the kidneys adequate scope and allowing for excessive loads of nitrogen and electrolytes. Adding this figure to that for the extra-renal loss, we obtain a total of 2,500–3,000 c.c. for the normal, basic water requirements. The daily requirements of salt cannot be calculated on this same basis, but there is general agreement that 5 grammes per day constitute an adequate allowance, and that the basic requirements are probably met by only 2 grammes.

In normal health there is, of course, a considerable daily variation in the intake, but the body is kept in balance by the kidneys, which excrete or retain water and salt as necessary. One of the most remarkable properties of the kidney is its extreme flexibility of action, and its power of differential control of water and salt excretion. However, it is just this flexibility of kidney function which is impaired after operation, when the kidneys are converted into comparatively rigid organs, becoming temporarily incapable of making the necessary adjustments to deal rapidly and adequately with varying loads of water and salt. This phenomenon is immaterial after operations of lesser magnitude, but becomes important when, owing either to the nature or magnitude of the operation, fluids must be given intravenously. There is some evidence that during the first twenty-four hours after operation there is a specific inability of the kidneys to excrete water, but this rigidity of kidney function essentially affects its powers of excreting sodium. If, during three to four days after operation, excess sodium (salt) is given, this sodium will be retained in the body, and with it water. If this excess administration is gross generalized œdema may develop, and with lesser degrees of retention there will be localized œdema in wounds and at suture lines, which may cause many undesirable complications. So during this period the intravenous administration of both water and salt should be carefully controlled, the basic ration being 4–5 pints of glucose solutions and 1 pint of normal saline per twenty-four hours: most certainly the whole intake should not be given as normal saline.

This temporary inability of the kidneys to excrete salt normally has another important result namely that during the post-operative period the urinary concentration of chlorides has not its usual significance. During this period a lowered urinary chloride concentration does not necessarily imply salt lack, and must not be taken *per se* as evidence for increasing the salt intake.

If, in addition to those by the normal route, there are abnormal losses from the body, then further fluid must be given. In abdominal and proctological cases these abnormal, excess losses arise almost invariably from the alimentary fluids, as in cases of paralytic ileus, or from fistulæ such as an ileostomy. Two well-known points in connexion with these fluids need emphasis. First, their enormous volume: normally some 8·5 litres of fluid per day are secreted into the alimentary tract, so that losses from this source can lead to rapid and severe dehydration. Secondly, these secretions consist not of water but of electrolyte-containing fluids: their total electrolyte content approximates to that of plasma, but the ratio of sodium to chloride varies considerably in the different fluids. Thus in gastric juice Cl is markedly in excess of Na, whilst the reverse holds true of pancreatic juice, and in the intestinal secretions themselves there is a less marked, but definite excess of Na over Cl. This varying loss of either sodium or chloride is important in that it can lead to disturbances in the acid-base balance, but too much attention can be focused on this disturbance. Save in exceptional circumstances this varying loss can be ignored, and the losses replaced by normal saline, as the kidneys will excrete the ion in excess. Studies carried out on a case of pancreatic fistula in which the loss was replaced in this way showed that the fistula fluid contained Na 155 m.Eq/L and Cl 57 m.Eq/L, whilst the urine concentrations were Na 66 m.Eq/L and Cl 181 m.Eq/L.

This method of volume-for-volume replacement can, of course, only be applied to cases in which the losses are observed. Essentially its efficacy depends upon giving the kidneys adequate water with which to make the necessary adjustments. Accordingly it is most important that these patients should receive their normal basic intake, as described above, and further that the losses should be carefully measured so that the replacement may be accurate. If this is done repeated chemical analyses of the plasma, urine, &c., are not necessary: only simple observations of the urine volume, urine specific gravity, and either the hæmoglobin or hæmatocrit are necessary. Provided the urine volume approximates to 1,500 c.c. per day with a gravity not exceeding 1015, and that the hæmatocrit does not rise, it can be safely assumed that replacement is adequate. In these cases with excess loss of alimentary fluids there is also, of course, a considerable loss of potassium, which may well need replacement, but this problem is outside the scope of the present discussion, as is, also, the much more difficult problem of the treatment of an actually dehydrated patient, that is the replacement of pre-existing as opposed to observed losses.

(Slides showing the electrolyte exchanges of a severe case of paralytic ileus were shown, to illustrate the points discussed.)

Ileo-anorectal Anastomosis for Multiple Polyposis of Colon and Rectum

By Stanley O. Aylett, M.B.E., F.R.C.S.

The purpose of this short paper is to report a case of familial polyposis of the colon and rectum in which an anastomosis between the ileum and anal canal has been carried out by a new approach with resulting complete continence.

It may seem presumptuous to report the result of a single case but this condition is not a common one and its treatment cannot be considered to be standardized. Only by a correlation of the experiences of every surgeon, as Hoxworth and Slaughter have endeavoured to show, will we be able to achieve a recognized and planned treatment.

It is to Dr. Cuthbert Dukes that we owe so much of our knowledge of the familial nature of this disease, and although Lockhart-Mummery in 1918 carried out the first total colectomy for the condition in this country, Dukes' emphasis on the certainty of malignant degeneration in every case has stimulated all surgeons dealing with these cases to carry out the widest possible excisions.

To-day the most common form of treatment is to carry out a total colectomy, with restoration of continuity of the bowel by means of an anastomosis between ileum and recto-sigmoid. The operation is performed in one or two stages and the polypi in the remaining rectum are burned by repeated fulguration. The difficulty in controlling these dozens of polypi by such a method is apparent and Dye, Guptill, Ravitch and Bartlett are among the authors who have reported cancerous changes in rectums so treated. Moreover, the method is not free of the dangers of stenosis if the fulguration is of necessity extensive, and of perforation, which has also been reported.

The problem is complicated still further when our pathological colleagues report on preliminary biopsies taken from the rectum as being in a pre-malignant state. Are we in such cases to continue with our faith in colectomy and fulguration, without an accurate appreciation of the extent of the pre-malignant change or of the effective range of the latter treatment? Or are we to carry out an excision of the rectum as well and condemn our young patient to a permanent colostomy?

In an endeavour to avoid these alternatives Ravitch carried out animal experiments in which, following a colectomy, he stripped the mucosal lining of the remaining rectum, and through the fibromuscular tube thus left pulled down the terminal ileum, which had been severed close to the cæcum, and stitched it to the anal skin. The dogs had satisfactory continence following this procedure and he advocated its use in the human for such conditions as familial polyposis and ulcerative colitis. He reported subsequently a case so treated with a final good result and Devine and Webb added 2 cases in whom continence was excellent. The experience which Goligher reported in his Hunterian Lecture has been the exact reverse and both cases were completely incontinent, necessitating in one an ileostomy of a permanent character. He attributes the failure of the operation to the fact that the anal stump was of a length insufficient to record that amount of rectal sensation upon which continence partly depends (Goligher, 1951).

At a previous meeting of this Section I presented 4 cases upon whom I had performed an anastomosis between the upper pelvic colon and the anal canal. On another occasion I demonstrated a patient in whom the ileum had been anastomosed to the anal canal by a pull-through method. (Aylett, 1950, 1951). The patients were here and I think those who questioned them must have agreed that they were all fully continent, with the ability to know when they required their bowels to act, and with the capacity to delay that action at will for periods of up to an hour. I, therefore, cannot agree with Goligher's explanation of the incontinence resulting in his cases and I think another reason must be sought.

The operation of stripping up the rectal mucosa requires extensive stretching of the internal sphincter in order that the area of operation may be visualized and I believe that irretrievable damage may result to this structure following such a manœuvre. We know that if the sphincter is divided, as in Babcock's operation, accurate resuture at the end of the operation does not restore its full function and incontinence is a common end-result. It appears, therefore, that the internal sphincter is a structure of some delicacy, with the anatomy of which it is unwise to interfere, either by division or by excessive stretching to the diameter of 8 c.m. as is required by this operation of mucosal stripping.

The operation I am about to describe leaves the sphincter completely undisturbed and it is because of this that I believe subsequent continence has been complete.

The abdomen is opened through a left paramedian incision and the pelvic colon and rectum are freed as in Miles' abdomino-perineal excision. Crushing clamps are applied to the rectum low down and the latter is then severed, the upper end being brought out through a stab incision in the left iliac fossa (Figs. 1 and 2).

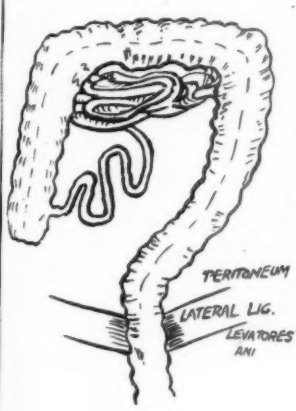

FIG. 1.

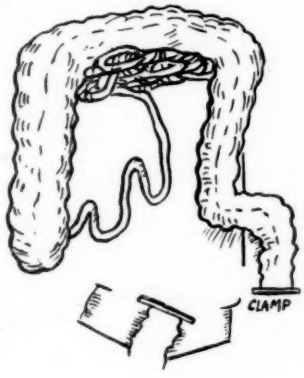

FIG. 2.

FIG. 3.

The terminal ileum is then cut across a few inches from the ileo-cæcal valve and both ends are closed. The proximal end is freed sufficiently to enable it to reach down to the depths of the pelvis (Fig. 3). The peritoneal floor is then re-established and is sutured to the wall of the ileum as it passes down into the pelvis, following which the abdomen is closed.

The patient is then turned on to his right side. A curved skin incision, extending from just behind the anus to the level of the sacro-coccygeal joint is made and the skin flap is dissected downwards (Fig. 4). The sacro-coccygeal joint is opened and the coccyx is removed after it has been severed from its lateral and anterior attachments. After division of the fascia of Waldeyer the terminal ileum and the remaining rectum are readily drawn out into the perineal wound. The pubo-rectalis portion

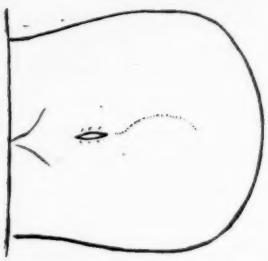

FIG. 4.

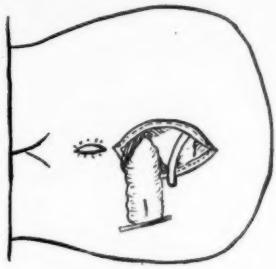

FIG. 5.

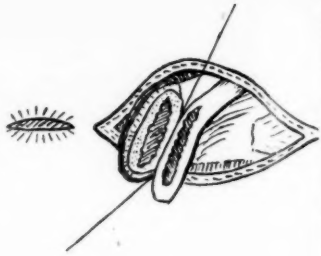

FIG. 6.

of the levatores ani muscle is then identified, curving round the anorectal junction in the form of a sling. The rectum is severed just above this level and the cut end is anastomosed to the ileum by an end-to-side anastomosis (Figs. 5 and 6) following which the wound is closed.

The patient upon whom this procedure was carried out developed complete continence and rectal control, although at the present time his bowels are opened 8 to 10 times in the twenty-four hours but I think that with the passage of time this frequency will diminish. The remainder of the colon was removed three months later.

It is true that the patient is left with a small ring of anorectal mucosa less than 1 cm. in depth, but this small cuff can be kept under periodic review. The possibility of polypi occurring in this region must be greatly less than when a large area of rectal mucosa is left behind, and the operation is a compromise between a Ravitch operation in which continence is uncertain, and ileo-rectosigmoid anastomosis, in which the large area of rectal mucosa remaining must be the source of constant concern with regard to malignant change.

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[*April* 3, 1952]

MEETING HELD AT THE LONDON HOSPITAL, E.1

The following Cases were shown:

Mr. J. E. RICHARDSON, Mr. A. J. WALTON and Mr. CLIVE BUTLER.
Hirschsprung's Disease Treated With and Without Surgery.

Mr. D. V. EVANS.
Right-sided Colitis Treated Surgically. Two cases.

Mr. H. ANNAMUNTHODO.
Carcinoma of the Rectum and Colon in a Patient with Chronic Colitis.

Mr. CLIVE BUTLER.
Familial Polyposis of the Colon with a Carcinoma of the Cæcum. Survival Eight Years After Right Hemicolectomy followed by Subtotal Colectomy.

Mr. HERMON TAYLOR.
(1) **Fistula-in-Ano Treated by Division of Sphincter Muscles and Immediate Suture.** (2) **Abdomino-perineal Excision of the Rectum and Hysterectomy with Ligature of One Ureter.**

Mr. CLIVE BUTLER.
Complete Rectal Prolapse in a Woman of 18 After Removal of a Cauda Equina Tumour. Successful Result Following Rectosigmoidectomy and Intra-Abdominal Repair.

Mr. B. J. FOWLER.
Unusual Accidents to the Large and Small Bowel.

Section of Pædiatrics

President—Professor A. G. WATKINS, M.D., F.R.C.P.

[*January* 25, 1952]

MEETING HELD AT ST. MARY'S HOSPITAL, LONDON

The following cases were shown:

Congenital Limitation of Joint Movement with Subsequent Progressive Muscular Dystrophy.—REGINALD LIGHTWOOD, M.D., F.R.C.P., D.P.H.

It is generally recognized that in the late stages of certain cases of muscular dystrophy, particularly when walking is no longer possible, secondary contractures may cause deformity, but such changes never occur early in the disease. That there exists a form of muscular dystrophy in which there is a limitation in the range of movement of certain joints recognizable at, or possibly before the onset of muscular dystrophy, is a separate observation not previously described. Therefore, two boys are presented in whom from earliest observation partial loss of active and passive joint mobility was recorded, though at their first examination they were not recognizably suffering from muscular dystrophy, and it was only later that this diagnosis became obvious. The first patient had a limitation in extension of both knees, also in abduction of the shoulders and in rotation and extension of the hips. The second had limitation in extension of knees only.

Case I.—I. F., born March 10, 1948. Only child of healthy parents. Normal pregnancy, normal fœtal movements. No evidence of hydramnios or oligo-hydramnios. Intelligence and early development apparently normal. Began walking at 1 year 8 months, with abnormal gait because of knee flexion. Arms never raised above the horizontal plane because of limitation of abduction of shoulder. Tumbled easily without putting hands out to break fall.

At 2 years 3 months referred to St. Mary's Hospital from an Orthopædic Hospital for pædiatric diagnosis: No wasting or weakness of muscles then noted. Peculiar gait attributed to knees being slightly flexed thus necessitating compensatory flexion of the hips and spine to maintain balance, but a film taken at this time shows that there was already a slight degree of weakness in the flexors of the hips. Joints: knees could be fully flexed but not fully extended, failure amounting to 10°–15° apparently due to some structural abnormality at the joints; its nature is not disclosed by X-ray, and a ligamentous abnormality is suspected. Hips showed slight limitation of extension of internal and external rotation. Shoulders showed nearly 90° restricted abduction. Other joints normal.

Nervous system: No loss of power detected except for slight weakness in quadriceps femoris. Cranial nerves: fundi normal. Reflexes: Biceps, triceps not obtained, knee and ankle jerks present and equal.

January 1951 (aged 2 years 10 months): Condition now recognized as muscular dystrophy. Gait had become waddling in character and pigeon-toed. Deep reflexes no longer obtainable. Subsequently the dystrophy progressed until he could not rise from the supine position without first rolling over and then holding on. The limitation of joint movement remained as before. Now considerable wasting of the limbs especially in their proximal segments. No pseudohypertrophy. No sensory changes.

January 1952 (aged 3 years 10 months): Weakness of the legs and thighs increased. No deep reflexes present. Plantar reflexes flexor. Cranial nerves: fundi normal, right pupil larger than left; both reacting to light and accommodation. No nystagmus, no squint. Intelligence normal.

A film was shown at the meeting demonstrating the evolution of the condition and the way in which the limitation of joint movements remained unchanged.

Case II.—M. B., born July 15, 1942. No significant family history: Parents not related; father's siblings (2 brothers, 1 sister); mother's siblings (1 brother, 2 half-brothers) all normal; patient has one normal sibling, female, born in 1944. Past history: Normal pregnancy and delivery. Normal development in infancy. Walking from 17 months with abnormal gait, pigeon-toed and knees slightly flexed. First seen May 1947 (4 years 10 months): knees could not be fully extended (defect of 10°–15°). Walked with gait similar to Case I. No evidence of muscular dystrophy noted. Five months later—knee and ankle jerks now unobtainable. Between 5 and 6 years became unable to walk upstairs and weakness of shoulder girdle then apparent, weakness progressive.

January 1952 (aged 9 years 6 months): Mentality and intelligence normal. Attends school for physically handicapped children. Cannot run or ascend stairs unaided. Parents consider condition now stationary. Gait pigeon-toed and undulating because of weakness of muscles round hip-joints. Forward stance, knees slightly flexed. Some scoliosis and valgus deformity of both feet. Cranial nerves, fundi normal. Sensation normal. Motor system: obvious wasting of upper and lower arms, pectorals and deltoids. Fasciculation in the wasted muscles of shoulder girdle. No wasting of trunk or abdominal muscles. Much wasting of glutei and thigh muscles, and moderate wasting below knees. All affected muscles weak, also moderate weakness of intrinsic muscles of hands and movements of wrists and elbows. Weakness of dorsiflexion and inversion of feet, the only normal power in the

limbs being eversion of the feet and plantar flexion. Deep reflexes absent. Plantar responses flexor. Superficial reflexes active.

A film was shown at the meeting demonstrating the character of this muscular dystrophy which mainly affects proximal muscle groups.

Comment.—There is a considerable resemblance between the muscular dystrophy which has appeared and then progressed in these two boys because it affects mainly the proximal muscle groups of all four limbs causing a waddling and a pigeon-toed gait. Fasciculation was observed in the wasted muscles of the shoulder girdle in the second case. Both of the patients showed limitation in the range of movement in certain joints which sign remained unchanged. This limitation was observed before the evidence of muscular dystrophy had clearly shown itself. A third patient, a male, with limitation in the extension of both keees from infancy and slight muscular weakness is also being observed. Since the above 2 patients and the film were shown, information concerning other similar patients has come to hand. The recognition of limitation in joint movement of the type described may perhaps be useful as a warning of the development of this type of muscular dystrophy. The condition in no way resembles arthrogryphosis multiplex (amyoplasia congenita).

The author acknowledges with thanks the help of Dr. C. H. Edwards, who confirmed the neurological condition described, and of Dr. P. Cardew who was responsible for the film. Mr. V. C. Snell kindly referred Case I.

Dolichosigmoid.—H. McC. Giles, M.R.C.P., D.C.H., and T. Pearse Williams, M.D., F.R.C.P.

Case I.—J. L., girl, aged 8 years. Well until one year ago when she began to soil her clothes with fæces, and has continued to do so until the present time. Motions are soft—not constipated. Often feels as if she wants to defæcate, but is unable to do so. On examination fæcal masses were palpable in the lower abdomen. Barium enema revealed a long pelvic loop of colon.

Case II.—M. W., girl, aged 5½ years. Constipated since early infancy. Bowels open twice weekly at first, decreasing to once weekly. Has been admitted to hospital on several occasions for treatment of exacerbations. Barium enemata in November 1950 and again in November 1951 show a long pelvic loop of colon. Her most recent admission to hospital was preceded by spurious diarrhœa for a month.

Comment by Dr. T. Pearse Williams.—The pelvic loop or sigmoid colon is proportionately of much greater length in the newborn infant and tends to lie well above the pelvic brim, up and to the right, even to the hepatic flexure (70%). By the process of differential growth common to other parts of the body and well exemplified by the prepuce it becomes in the adult a much smaller section of the colon. The long loop persists in adult life in about 2% of individuals only. In my opinion it is one of the factors to be considered in persistent constipation in the growing child and may be associated with fæcal impaction as in the case of J. L. or a developing idiopathic megacolon as in M. W. I cannot support the view that idiopathic megacolon is due to colonic inertia.

Presumed Dermatomyositis with Cardiomegaly, Trichiniasis and Epilepsy.—F. S. W. Brimblecombe, M.D., M.R.C.P., D.C.H., and M. Keech, M.D., M.R.C.P. (for Reginald Lightwood, M.D., F.R.C.P., D.P.H.).

P. C., boy born May 1946. Normal development.

1949, August (aged 3): Paddington Hospital with infective hepatitis and left otorrhœa. Normal-sized heart radiologically. Never fully recovered; listless and constipated with persistent anorexia, vomiting, hepatomegaly and weight loss, with muscle weakness preventing walking.

1950, March: Acute heart failure responding to digitalis (St. Mary's). All investigations normal apart from an increased blood pyruvic acid which remained unaltered after parenteral thiamin and co-carboxylase (Professor Sir Rudolph Peters) (Fig. 1).

May (Taplow Hospital): Cardiomegaly with generalized weakness of all voluntary muscles and of intestinal muscle producing a Hirschsprung picture.

June: Minimal pericardial effusion demonstrated by electrocardiography and cardiac catheterization (Dr. E. M. M. Besterman); three muscle biopsies showed a solitary trichinella cyst not more than nine months old (Professor J. J. C. Buckley) and a peculiar granular degeneration of muscle fibres (Dr. L. E. Glynn). Liver biopsy showed no evidence of amyloid or glycogen disease. All investigations were normal except for a raised blood pyruvic acid and a low fasting blood sugar and creatinine coefficient. There was no eosinophilia, muscle tenderness or rash, but the temperature and sedimentation rate were persistently elevated and a mild anæmia was unaffected by oral iron.

1951, August: The general muscle tone had improved sufficiently to allow of walking with axillary support and of riding the tricycle in the Physiotherapy Department. He was very bright and talkative and the constipation was fairly well controlled with daily laxatives and bi-weekly enemas.

October: Onset *petit mal* progressing to minor *grand mal* in November.

December (St. Mary's Hospital): An EEG was grossly abnormal, indicating widespread cerebral degeneration, but the C.S.F. was normal.

Present condition.—Mentally apathetic, marked muscle weakness and frequent attacks lasting half an hour with loss of speech and repeated fine tremor of lips and hands.

Comments.—This case presents considerable diagnostic difficulty. Low creatinine coefficient, low

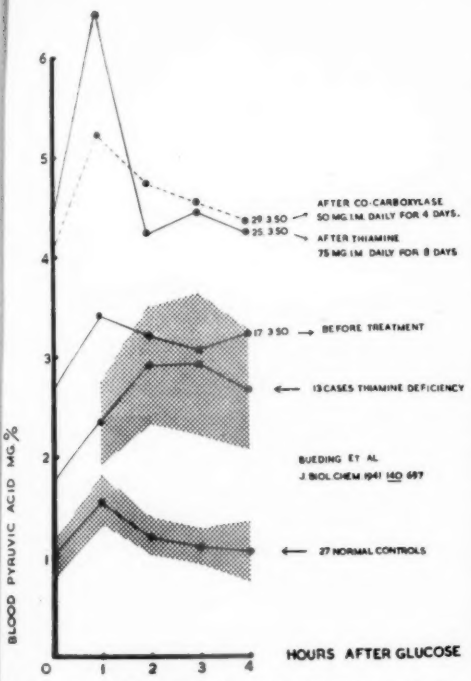

FIG. 1.—Blood pyruvic acid following glucose ingestion: (i) 17.3.50: no treatment; (ii) 25.3.50: after thiamin; (iii) 29.3.50: after co-carboxylase. Case P. C., aged 4 years.

FIG. 2.—Biopsy rectus abdominis muscle, showing marked granular degeneration and vacuolation of the striated fibres and proliferation of sarcolemmal nuclei. H. & E. × 250.

blood sugar and persistently raised blood pyruvate can occur in widespread muscle degeneration from any cause. The following reasons for cardiomegaly were excluded: (*a*) Congenital (by normal X-ray, March 1949). (*b*) Thiamin deficiency. (*c*) Glycogen disease (by liver biopsy).

The remaining possibilities are dermatomyositis, or trichiniasis.

The points in favour of dermatomyositis are the prolonged generalized muscle weakness, the cardiomegaly and heart failure (Keil, 1940; Roch *et al.*, 1946), and the histological appearance of the muscle (Fig. 2). Against it is the development of epilepsy and cerebral degeneration.

The points in favour of trichiniasis are the biopsy containing the parasite, the generalized muscle weakness, myocardial involvement, epilepsy (Dandy, 1947), and cerebral degeneration (Most and Abeles, 1937; Gordon *et al.*, 1935). Against it is the lack of eosinophilia (although proved cases have been reported without this blood change); the absence of history of muscle invasion and the chronic course with no calcified cysts radiologically two and a half years after infection. Most cases have an acute onset with either rapid recovery in four to six weeks or a fatal termination. However, the age of the parasite would coincide with the acute illness diagnosed as infective hepatitis which was followed by progressive muscle weakness, constipation and hepato-cardiomegaly. There is no mention of jaundice in the literature on dermatomyositis or trichiniasis, although prolonged constipation may occur in the latter (Gould, 1945).

Conclusion.—It is impossible to be dogmatic about the diagnosis but this is probably a case of dermatomyositis with intercurrent trichiniasis producing the cerebral manifestations.

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Recovery from a Large Frontal Lobe Abscess in Infancy in a 6-year-old Child.—D. L. B. FARLEY, F.R.C.S.

When aged 3 months he was admitted to the Neurosurgical Service of the Manchester Royal Infirmary with a history of left cavernous sinus thrombosis, treated at the Bolton Royal Infirmary one month previously.

On examination.—The head was grossly enlarged and the fontanelles were tense and bulging. There were no abnormal physical signs in the central nervous system, neither motor nor sensory.

W.B.C. 13,800.

Cerebrospinal fluid: Pressure 400 mm. C.S.F.; no block. Protein 120 mg.%. 400 cells (polymorphs and lymphocytes) per c.mm. Sterile.

X-ray chest and X-ray skull negative.

Diagnosis.—This lay between a frontal lobe abscess and chronic meninigitis associated with external hydrocephalus.

Needling of the brain (24.4.46).—Diagnostic needling through the anterior fontanelle proved the presence of an enormous left frontal cerebral abscess. 145 c.c. of pus were obtained. This grew *Staph. aureus*, penicillin sensitive. Penicillin and Thorotrast were inserted into the cavity (Fig. 1).

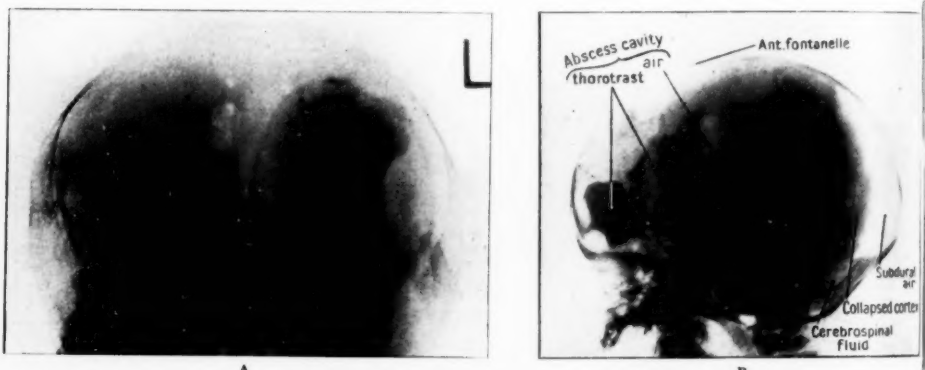

FIG. 1.—(A) Postero-anterior and (B) lateral views taken 24.4.46, immediately after the first aspiration and insertion of 2 c.c. of Thorotrast into the cavity. The abscess apparently fills the whole of the left frontal lobe extending back to the origin of the motor cortex.

FIG. 2.—Lateral view taken on 25.1.52 showing the scar in the left frontal lobe.

Patient was treated by systemic penicillin and by six aspirations and insertion of penicillin into the cavity.

Progress.—Continual improvement. Discharged 6.6.46 with a left VI nerve palsy as the only abnormal sign.

X-ray (28.10.46): This showed almost complete obliteration of the cavity. These films were lost but a recent X-ray (25.1.52) shows almost exactly the same picture (Fig. 2).

In 1947 for two months had frequent minor convulsions which disappeared spontaneously. There have been no major convulsions.

Speech was very retarded until the age of 4, when it proceeded rapidly. Development was normal apart from this.

Present condition (aged 6 years).—The child is fit and well and is of normal intelligence for his age.

Examination of the cranial nerves shows slight asymmetry of the face owing to a suspicion of a right facial weakness. His optic discs are normal and there are no other abnormalities. Examination of limbs shows no abnormality of tone, power or reflexes. Both plantar responses are flexor. There is some abnormality of the left maxillary margin and there appears to have been a slight retardation of growth.

Comment.—There were no constitutional effects of this abscess. The only physical signs were those of enlargement of the head and a sterile but abnormal cerebrospinal fluid. Diagnosis was made by needling. Treatment was by aspiration and penicillin insertion. Recovery is, to all intents and purposes, complete.

Congenital Urethral Obstruction Presenting as Ascites in the Neonatal Period.—J. A. DAVIS, M.B., B.S. (for URSULA JAMES, M.R.C.P.).

I. P., male, aged 8 months.

History.—Mother, normal pregnancy and delivery. No abnormality noted in baby aged 1 week. When aged 3 weeks he suddenly developed anorexia, drowsiness and abdominal swelling, and was admitted to hospital. No frequency of micturition; good stream of urine.

On examination.—Drowsy, dehydrated baby with gross abdominal swelling but no œdema. Signs of ascites. Kidneys and bladder impalpable.

Investigations.—Paracentesis yielded ascitic fluid, after which a suprapubic tumour, previously not noted, was felt. Laparotomy revealed a grossly dilated and hypertrophied bladder, and catheterization an obstruction in the posterior urethra. This obstruction was broken down from both directions and an intravenous transfusion given.

Subsequent progress has been satisfactory and there are no signs of urethral obstruction or renal inadequacy. Weight gain normal. Hb 80%; Blood urea 15 mg.%; I.V.P. slight dilatation of pelves and ureter more marked on right. Right kidney palpable but bladder small.

Comment.—Of the many cases of congenital urethral obstruction reported, only a small proportion were diagnosed in infancy, but of these many presented with abdominal swelling. Of the possible explanation of the ascites, the most attractive is that the fluid is filtered through a thin area of bladder wall. Other possibilities are actual rupture of the bladder or a general effect of impaired renal function.

Idiopathic Hypercalcæmia with Failure to Thrive: Nephrocalcinosis.—REGINALD LIGHTWOOD, M.D., F.R.C.P., D.P.H.

R. E., male, born 3.5.51, birth-weight 8 lb. 4 oz., only child, breast fed for one month, then half-cream Cow and Gate, then full-cream Cow and Gate. From 1 month old the daily intake of vitamin D had been approximately 1,400 i.u.—not believed to be a "toxic" dose. At about the end of the third month appetite failed, he vomited persistently and became constipated. Failure to thrive continued and at 6 months he was admitted to Farnborough Hospital, Kent, where he was treated for "infection of urinary tract" but the symptoms did not abate; therefore *renal acidosis* was considered and he was transferred to my care at 8 months, weighing 12 lb.

Examination (14.1.52).—Somewhat dehydrated, considerably wasted, alert, normal complexion, some hypotonia, no other abnormal physical signs.

Urine: Acid, trace of protein, occasional W.B.C., numerous epithelial cells, no casts; "clean" specimens were examined and cultured at weekly intervals—2 sterile, 1 light mixed growth of *Proteus vulgaris* and *B. coli.*

After correction of dehydration chemical examination of the blood showed the following normal findings: Plasma chlorides 619 mg.%. Plasma bicarbonate 53 vol. CO_2%. Serum sodium 326 mg.%, serum potassium 15·4 mg.%, blood inorganic phosphorus 3·8 mg.%, alkaline phosphatase 9·4 units (King-Armstrong).

The symptomatology had suggested *renal acidosis* but the above results and the acid reaction of the urine excluded this diagnosis. In similar cases according to experience hypercalcæmia and slight urea retention may be found and in this patient the tests showed serum calcium 13·9 mg.%, blood urea 46 mg.%. Furthermore a minority of infants with this clinico-chemical picture have X-ray evidence of renal calcification of the medullary type and it was successfully demonstrated in this case.

Course.—During the first two weeks of observation the hypercalcæmia varied between 13·0 mg.% on January 22 and 14·2 mg.% on January 29. On January 23 the sedimentation rate was 48 mm. in one hour (Payne's micro-method). A small blood transfusion was given to correct anæmia (R.B.C. 2,810,000; Hb 60%) and the hæmoglobin rose to 95%. By February 13, a month after coming under observation, the appetite had improved and the weight was increasing, serum calcium 10·6 mg.%; blood urea 58 mg.%; anorexia and occasional vomiting continued but the condition was gradually improving. The patient is still under observation.

During the last two years at the Hospital for Sick Children, Great Ormond Street, we have been studying the course and clinical features of a number of similar cases, provisionally named *idiopathic hypercalcæmia.* In these cases spontaneous recovery has usually occurred and the prognosis for the infant now reported is considered to be good. At the same centre Dr. W. W. Payne and his colleagues are studying the chemical nature of the condition and it is to him that I am indebted for the chemical data quoted.

Two Papers were read, (1) "Neonatal Response to Carbon Dioxide", by **K. W. Cross,** M.R.C.P., and **J. M. D. Hooper,** B.M., B.Ch., and (2) "The Effect of Testosterone and Casidrol in the Feeding of Premature Babies", by **Ursula James,** M.R.C.P.

Both papers are to be published elsewhere.

The following Cases were also shown:

Ganglioglioma.—Dr. J. W. G. TUTHILL (for Dr. R. LIGHTWOOD) (previously shown to the Section on November 23, 1951, by Dr. M. E. PENDLETON. See *Proc. R. Soc. Med.*, 1952, **45,** 223.)

Two Cases of Fanconi's Anæmia.—Drs. E. NEUMARK, R. LIGHTWOOD, J. D. L. REINHOLD and CEDRIC CARTER.

Four Cases of Adipose Gynism and Adipose Gynandrism (Two in Siblings).—Drs. J. A. DAVIS and S. LEONARD SIMPSON.

Two Cases of Infantilism.—Drs. J. A. DAVIS and S. LEONARD SIMPSON.

Two Atypical Cases of Cerebral Palsy.—Dr. URSULA SHELLEY.

Achondroplasia with ? Allergic Eosinophilia.—Dr. Z. MONCRIEFF.

Vitamin-D Resistant Rickets.—Dr. J. E. JELINEK (for Mr. V. H. ELLIS).

Persistent Visible Peristalsis in Sigmoid Colon.—Mr. DESMOND FARLEY (for Professor C. ROB).

Localized Cystic Disease of Lung.—Dr. M. COX (for Dr. F. S. W. BRIMBLECOMBE).

Renal Acidosis with Hypercalcæmia.—Dr. J. W. G. TUTHILL (for Dr. R. LIGHTWOOD).

The following Demonstrations were presented:

(1) **Respiratory Tracings of Newborns.**—Drs. K. W. CROSS and J. M. D. HOOPER.

(2) **Demonstration of Anoxic Impulses in Carotid Sinus Nerve in Newborn Animals.**—Drs. K. W. CROSS and J. L. MALCOLM.

(3) **Phrenic Nerve Stimulator.**—Drs. K. W. CROSS and P. W. ROBERTS.

(4) **The Oxygen Concentration in the Queen Charlotte Tent and Laboratory Thermal Equipment Limited Incubator.**—Dr. J. M. D. HOOPER.

(5) **The Unmeasured Loss of Electrolytes in Balance Studies.**—Dr. T. STAPLETON.

(6) **Clinical and Bacteriological Aspects of Pyogenic Meningitis.**—Drs. J. A. DAVIS, T. STAPLETON and G. T. STEWART.

(7) **Experience of Tuberculous Meningitis in Children in the St. Mary's Hospital Pædiatric Group.**—Drs. F. S. W. BRIMBLECOMBE and J. A. DAVIS.

(8) **Observations on the Ætiology of Bronchiectasis.**—Drs. F. S. W. BRIMBLECOMBE, J. A. DAVIS and J. W. WELLS.

(9) **Electrolyte Excretion in a Case of Nephrosis after Mersalyl and After ACTH.**—Dr. F. S. W. BRIMBLECOMBE.

(10) **The Radiology of Abdominal Emergencies in the Newborn.**—Dr. E. ROHAN WILLIAMS.

(11) **The Bacteriology of Sinusitis.**—Drs. S. E. BIRDSALL and G. T. STEWART.

(12) **Epidemiology of Sonne Dysentery.**—Dr. G. T. STEWART.

(13) **A Case of Fatal Bronchopneumonia associated with An Unusual Strain of Agglutinable *Bact. Coli*.**—Dr. G. T. STEWART.

(14) **Neonatal Record Card, St. Mary's Hospital.**

(15) **Cystic Agenesis of Brain with Hamartoma of Optic Chiasma.**—Dr. D. M. PRYCE.

(16) **Transillumination Photographs of Ossifying Sternum.**—Dr. D. M. PRYCE.

(17) **Liver Tumours in Childhood.**—Drs. H. H. G. EASTCOTT, E. NEUMARK, J. I. PUGH and A. WYNN WILLIAMS.

(18) **The Note-Books of the Late Dr. F. J. Poynton.**—Dr. J. G. BATE.

(19) **The Radiology of Nephrocalcinosis.**—Dr. J. W. WELLS.

(20) **Cold Vapour Nebulizer (Denton-Smith).**—Dr. J. W. G. TUTHILL.

(21) Medical Illustration (Mr. P. J. FISKE; Clinical Photographer, Canadian Red Cross Memorial Hospital, Taplow) **(i) Rheumatic Fever—Clinical and Pathological Aspects. (ii) Miscellaneous Photographs—The Collagen Diseases and Still's Disease.** (The Special Unit for Juvenile Rheumatism, Canadian Red Cross Memorial Hospital, Taplow. Director: Dr. E. G. L. BYWATERS).

(22) **Exhibition of Clinical Photographs** (Shown jointly by Pædiatric, Photographic and Radiological Departments of St. Mary's Hospital), arranged by Dr. H. MCC. GILES.

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President—JOHN WILKIE, M.Sc., M.B., F.F.R., D.M.R.E.

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The Place of Radiotherapy in the Treatment of Simple Skin Conditions

PART I

By ALEXANDER A. CHARTERIS, M.B., Ch.B., D.P.H., F.R.F.P.S.G., F.F.R.

Director of Radiotherapy Department and National Radium Centre, Western Infirmary, Glasgow

WE believe that the treatment of simple skin conditions by radiotherapy will be better carried out where there is close co-operation between dermatologist and radiotherapist and also that accurate records and thorough follow-up are essential. In the Western Infirmary Dr. James Sommerville and I hold a combined clinic twice weekly in the Radiotherapy Department and this association has been a guide in finding what are the most efficacious methods. While the dominant partner must be the dermatologist, the radiotherapist provides satisfactorily calibrated apparatus and sees that prescriptions are accurately carried out, at the same time advising on many biological problems and dealing with the question of protection. His experience of the long-term effects of irradiation is also valuable in planning treatment for the more chronic or recurrent cases. This is important since the treatment of simple skin conditions may be more dangerous than that of malignant ones because the absence of reaction and of gross skin changes may lull the unsuspecting into continued efforts, the ultimate effects of which will not be obvious for many years. Skin atrophy, damage to skin appendages, chronic radio-dermatitis and even malignant change can all be seen where certain limits have been transgressed, though such complications should not occur nowadays. Apart from such changes, unwise treatment may cause arrested development to certain tissues in young people, and I have seen examples of this in the teeth and the mammary gland, while possible effects upon the epiphyses and upon the eye have also to be borne in mind.

Of the efficacy of radiotherapy in simple skin conditions there can be no doubt, but the rationale of its action is not always clear, although such processes as the clearing of cellular infiltration and the treatment of certain infective conditions can be understood fairly well. Radiotherapy is not routinely applied to simple skin conditions but only after careful consideration of the use of normal dermatological treatment methods, and where it is used we have tried to assess critically the effects of varying kilovoltages, dose-levels and fractionation. The main conclusions will be stated by Dr. Sommerville. For completeness I shall mention briefly three conditions with which my department has had a good deal of experience.

The first is the common *plantar wart*. During the years 1947–1949 we saw 286 cases. We do not necessarily apply radiotherapy routinely, but, in fact, the majority of these cases had to be treated either by radium application—delivering 1,300 r incident dose in six hours—or by X-ray therapy, giving 1,200 r in three doses over a week, or less often 1,000 r in a single dose, mostly at 85 kV. There seems to be no difference as regards the efficacy of these two methods, satisfactory results being obtained in between 80% and 90% of cases. We have never observed any unpleasant effects and believe this is due to our inviolable rule of giving one treatment or course of treatment only. Two applications are not only useless but dangerous, and cases which have failed must be dealt with on other lines.

54 cases of *keloid or hypertrophic scar* were treated during the years 1947–49, only 11 being in association with surgery, and the remainder having radiotherapy alone. Most cases have X-ray therapy at 85 kV. with doses of 300 r monthly for three or perhaps four doses, the spacing between the last two being determined by response. Radium moulds have been used in a smaller number of cases, the dose-level being 500 r at each exposure and the results have been very good. Only 3 cases of the total showed a poor result, 7 were fair and the rest were regarded as satisfactory. A keloid of any size in an exposed site will generally call for more than the treatment required merely to change a raised scar to a flat one, and the present policy is therefore swinging over to pre-operative and post-operative treatment.

The cavernous and mixed varieties of *hæmangiomata in children* are eminently treatable. We are aware of the school of thought which prefers to leave these alone on the grounds that many will regress spontaneously, and also because of the alleged risk of tissue damage from radiotherapy, but we believe in giving treatment for cosmetic and psychological reasons, as well as to avoid risks of injury with consequent sepsis and gross scarring. The fact that properly planned radiotherapy entails no reaction and no risk of either skin change or damage to developing tissues seems to render this point of view rational. Treatment is either with radium moulds at 0·5 cm. distance and dose at skin of 400 r, repeated in a month, and thereafter as regression dictates: or less often with X-ray therapy at

85 kV., the individual doses being about 300 r. The advantages of the moulds are great since the child is not alarmed and there is no question of the mother or the staff being exposed to radiation during X-ray treatment, and this is our method of choice, giving excellent results. During the years 1947–1949 564 cases were treated, many having multiple lesions, and failure to achieve regression was noted in only 11 cases. For the rest, about 20% had a good result, the remainder being classed as "excellent", where no trace of the lesion could be seen, or "very good" where there was very little trace. No damage or skin change, apart from that due to previous ulceration or previous scarring, has been observed.

In conclusion, I should like to acknowledge my gratitude to Dr. Sommerville for his co-operation and help. Both he and I acknowledge our thanks to my staff, who have analysed the large number of case-sheets and produced therefrom the concrete facts and figures.

Part II

By James Sommerville, M.B., F.R.F.P.S.(G.)

The McCall-Anderson Lecturer on Dermatology, The University, Glasgow; Senior Physician-Dermatological Unit, The Western Infirmary; Senior Physician for Diseases of the Skin, The Southern General Hospital; Honorary Consultant Dermatologist, The Royal Hospital for Sick Children, Glasgow

In the new X-ray unit of the Western Infirmary, Glasgow, responsibility for treatment prescribed in simple skin conditions is still maintained by a system of joint skin/X-ray therapy sessions.

This system had been introduced by Dr. J. Struthers Fulton. The senior dermatologist is responsible for the suggested line of therapy, the radiotherapist undertaking to see it carried out. This scheme has worked admirably and harmoniously with benefit to both sides. Schemes of treatment variations and modifications have been from time to time suggested by both.

This contribution is drawn from conclusions based on work carried out over the years 1947–1949 and, in some cases, 1950. No attempt is made to cover the whole field of simple skin conditions. Dr. Charteris has already referred to some of the benign hyperplasias. Any conditions omitted are left out because their numbers are too small to draw satisfactory conclusions from them. It should be stressed that radiation therapy is only one weapon in the therapeutic armamentarium of the dermatologist. It is a weapon which should never be used lightly, and only when other treatments general and local have had adequate trial. In simple skin conditions it should not be used until one is satisfied that the desired result cannot be achieved by other measures. This attitude will explain the paucity of numbers in some of the following tables, which deal with each section.

Dr. Charteris has described the types of irradiation used.

Tables I and II show two methods used to achieve temporary epilation in sycosis barbæ, the five-field method being the one at present used as more uniform results are obtained.

Table I.—Epilation Scheme (Original Method)

	Size diam. in cm.	F.S.D.	Filter	kV.	mA.	r/min.
Left facial ..	10	25 cm.	0·25 Cu 1 Al	140	8	32
Right facial ..	10	25 cm.	0·25 Cu 1 Al	140	8	32
Upper lip and chin	8	25 cm.	1 Al	85	8	37

Table II.—Epilation Scheme (Present Method)

	Size diam. in cm.	F.S.D.	Filter	kV.	mA.	r/min.
Left facial ..	10	25 cm.	0·25 Cu 1 Al	140	8	32
Right facial ..	10	25 cm.	0·25 Cu 1 Al	140	8	32
Upper lip and chin	8	25 cm.	1 Al	85	8	37
Right cervical ..	10	25 cm.	1 Al	85	8	38
Left cervical ..	10	25 cm.	1 Al	85	8	38

Table III explains the results in 51 cases of sycosis barbæ—these cases being subacute to chronic with an average duration of thirty-three months. The age distribution is striking. The best response occurs in those with satisfactory temporary epilation, about 33% being cured. For fractionated therapy the only claim that can be made is of some temporary improvement, as although all cases benefited initially to some extent, 44·4% relapsed.

TABLE III.—SYCOSIS BARBÆ (51 CASES)

Duration: 2/12–10 yrs. : average 33 months

Age incidence in years	−20	20+	30+	40+	50+
Number	1	30	9	9	2

	No.	Well	%Gp.	Imp.	%Gp.	Unch.	Rel.	% Imp.
Epilation—Satisfactory	34	11	29·3	23	68·3	—	3	10·7
Epilation—Unsatisfactory ..	7(B)	1		5		1	—	—
Fractionated (A)	10	1	10	9	90	—	4	44·4
Total	51	13		37		1	7	

1..3 × 50..Stopped due to impetigo

(A) 3..6 × 50..1 well

3..8—12 × 50

1..8 × 75

1..5 × 100

1..3 × 120

(B) Unrelated to age or duration

Table IV relates to 8 cases of furunculosis—in which only 2 had a single lesion, both of which were treated by the single 200 r dose. All of the cases are shown in detail—no matter what type of irradiation was used, and whether small doses were used over a longer period or larger doses over a shorter period the results were uniformly good.

TABLE IV.—FURUNCULOSIS (8 CASES)

Age	Site	Duration	kV.	Dose r	Time	Total	Response
42	Neck	14 years	85	5 × 25 4 × 25 6/12	16 D 3 W	225	Good
32	Neck	4/12	85	6 × 25	5/12	150	G
19	Neck	1 year	140	12 × 25	11/52	300	G
56	Axillæ	1/12	140	4 × 50	2/52	200	G
35	Axilla	1/12	140	200	S.D.	200	G
46	Elbow	1/12	200	200	S.D.	200	G
24	Axilla	2/12	200	6 × 75 6 × 25 1/12	5 W 5 W	600	G
29	Axilla	5/12	200	6 × 75	5 W	450	G

All cases were male

Table V describes the results in 9 cases of the chronic indolent condition of paronychia. In these 140 kV. was preferred to achieve greater depth effect. In two-thirds of the cases treated the results were good, and the table shows that the dose level required is round about 600 r (*see* p. 410 for key to Tables V–XIV).

TABLE V.—PARONYCHIA (FINGERS) (9 CASES)

Result	Sex M	Sex F	Age incidence	Duration	140 kV. — 300 r	140 kV. 300 r to 600 r	140 kV. 600 r
Good	—	6	36–56	5/12–6	4 × 75 — 1	4 × 75², 4 × 150 — 3, 4 × 75²	12 × 50 — 2, 5 × 200 (Lv)
Moderate	1	1	U	4/12–10	{8 × 25, 3 × 30} — 1	8 × 25² — 1	—
Poor	—	1	U	1/12	5 × 60 — 1	—	—
Total	1	8	36–56	1/12–10	3	4	2

Table VI concerns 72 cases of acne vulgaris. It should be noted that 59 of these, shown as cured or improved, occur in the dosage rate of 100–150 r weekly. This skin condition, in which hereditary, endocrinous, dietary, and occupational factors play such an important and varying part well illustrates the futility of expecting X-ray therapy to act as a charm. *Only* 18·05% are claimed as cured and these occur only in the 100–150 r dosage rate. In spite of this, fractionated X-ray therapy offers a very great chance of quite distinct improvement when all other measures seem to fail. (Coloured transparencies in support of this conclusion were submitted.)

TABLE VI.—ACNE VULGARIS (72 CASES)

Result	Sex M	Sex F	Age incidence 15	20	25	30	35	40	45	X-ray dosage 25	50	75	100	150	200	250	%
Well	5	8	—	3/2	1/4	1/2	—	—	—	—	—	—	2×9, 6×12, 1×15, 2×18 — 11	5, 2, 7	—	—	18·05
Improved	35	22	—/2	17/9	13/3	4/7	—/1	—	1/—	1	5+, 6, 10	21, 6, 6, 4, 12, 9	6, 37	5.6.7, 8.9.10, 9, 11, 12, 12	1	1	79·16
Unchanged	1	—	—	1	—	—	—	—	—	—	—	—	1	—	—	—	1·385
Worse	1	—	—	1	—	—	—	—	—	—	—	—	1	—	—	—	1·385
Total	42	30	2	33	21	14	1	—	1	1	4	4	50	11	1	1	100

37 improved cases treated with 100 r: 2 × 3, 1 × 4, 1 × 5, 1 × 6+, 2 × 8 — (1 × 9), 3 × 10 — 10; 13 × 12, 4 × 14, 1 × 15, 3 × 16 — 6 × 18 — 27

In Table VII consideration is given to the results of 12 cases of localized hyperidrosis—a condition governed by so many conditions, including focal infection, contact reactions, anatomical maladjustments and nervous control—all factors which, if possible, must be controlled before considering X-ray therapy. The condition is not only distressing but can actually be incapacitating. The scheme of X-ray treatment required has formed the basis of some considerable difference of opinion between

Dr. Charteris and myself. The results show that the best results have been achieved with 140–220 kV. and with a dosage rate at or about 800 r. Treatment is now standardized at 300 r once every three weeks up to a total of 900 r.

TABLE VII.—HYPERIDROSIS (12 CASES)

Result	Sex M	Sex F	Age incidence	Duration (years)	kV. 85 500 r to 800 r	kV. 85 800 r +	kV. 140 500 r to 800 r	kV. 140 800 r +	kV. 220 — 500 r
Good	2	5	17–48	Many years	—	1 850 (5) 18/12	800 (2) 1/12 500 (4) 4/52 3 750 (10) 9/52	950 (2) 12/12 2 2,000 (6) 2 yr.	1 300 (1)
Moderate	1	—	51	Many years	—	—	—	—	1 400 (1)
Slight	1	1	21–31	17	—	—	500 (1) 2 500 (1)	—	—
Unchanged	1	1	19–25	1½	500 (1) 2 500 (2)	—	—	—	—
Total	5	7	17–51	—	2	1	5	2	2

Table VIII assesses the results obtained in 36 cases of pompholyx, a recurrent troublesome condition governed by almost the same factors as in the last group. In all cases the treatment was fractionated, and it would seem that better results are obtained with a higher kV. and with larger rather than smaller fractions. Today 140 kV. giving either 3 × 125 r over four weeks or 4 × 75 r in three weeks is regularly prescribed.

TABLE VIII.—POMPHOLYX (36 CASES)

Result	Sex M	Sex F	Age incidence	Duration	kV. 85 — 50	kV. 85 75 +	kV. 140 — 50	kV. 140 75 +	kV. 200 100	Relapse	Additional therapy	Response
Cured ..	7	6	10–42 / 28–56	6/12–15	4.6.7. 3	—	6 *	4.4.4. 3 **	4 4 1	*3 **1	4 × 30	Imp.
Improved	16	6	22–55 / 25–49	5/52–5	7 7 4 8 8	1 4×150	5×9 (8×25) 11 5×8	6.6.6 4 4 6 4×100	—	7/11 9 2/6	5	4 Imp. 1 C.
Unchanged	—	1	— / 53	4	—	—	1 7+6	—	—	—	—	—
Total	23	13	10–42 / 25–56	5/52–15	7	1	18	9	1	13	—	—

Table IX deals with that distressing condition pruritus ani, 8 cases of which come under review. Again these cases were submitted to radiotherapy after all potential causes had been dealt with and other measures had failed. Here it is evident that a higher kV. seems more effective and only after a dose level of 400–600 r has been reached.

TABLE IX.—PRURITUS ANI (8 CASES)

Result	Sex M	Sex F	Age incidence	Duration	kV. 85	kV. 140
Cured	2	2	55–60	1/12–3	$\frac{6\times50+4\times25}{2}$ 3×400 (warts)	$\frac{4\times150}{2}$ $3\times50+3\times150$
Improved	—	2	42	8/12	—	$\frac{2}{6\times50}$
Unchanged	2	—	30–45	15/12–30/12	$\frac{5\times50}{2}$ 8×25	—
Total	4	4	30–60	1/12–3	4	4

In Table X are seen the results obtained in 21 cases of lichen simplex chronicus or what may be called circumscribed neurodermatitis where, it must be recalled, one is dealing with a thickened epidermis and a fair degree of infiltration in the corium. It is clear that better results would seem to have been achieved with 85 kV. in 75–150 r dosage given fortnightly in from 4–6 treatments. This is a little unexpected. One would have thought that 140 kV. offered better prospects. At this kV., however, the numbers are too small to be of any comparative value.

TABLE X.—LICHEN SIMPLEX CHRONICUS (21 CASES)

Result	Sex M	Sex F	Age incidence	Duration	85 kV. 25–50 r 6 Weekly	85 kV. 75–150 r 4–6 Fortnightly	140 kV. 50 r 5–6 Weekly	140 kV. 150 r 4 Fortnightly
Cured	1	6	15–43	3/12–7	2	5	—	—
Improved	6	6	19–76	2/12–20	5	5	1	1
Unchanged	—	2	40–56	10–15	1	—	1	—
Total	7	14	15–76	2/12–20	8	10	2	1

Table XI might have been slumped together with the last table—but these 29 cases represent chronic infective or irritant patches which, due to scratching and chronicity, have become thickened and infiltrated. Here the best results have been obtained with comparatively higher doses in both 85 and 140 kV. and today 3 × 125 r over four weeks is regularly suggested.

And finally Tables XII to XIV evaluate the X-ray treatment of the dermatitic group. These have been assessed in three tables according to their dermatological nomenclature. Although possibly they could easily be classed together, nevertheless there is an ætiological difference, albeit the pathological picture is essentially the same.

Table XII is composed of 38 cases of dermatitis infectiosa or infectious eczematoid dermatitis, and comprises examples at all stages of subacute to chronic activity. All showed response to X-ray therapy and the best results occur in the higher dose level of both 85 and 140 kV.

Table XIII is made up from 23 cases of contact or irritant dermatitis again in all stages of subacute to chronic activity. The preponderance of cases is female, and most of these are associated with simple soap and water irritation. This probably is the reason for the failures and relapses noted in this group. There is once more the same indication of therapeutic response to higher doses in both 85 and 140 kV.

TABLE XI.—DERMATITIS LICHENOIDES (29 CASES)

Result	Sex M	Sex F	Age incidence	Duration	85 kV. — 50 r	85 kV. 75 r +	140 kV. — 50 r	140 kV. 75 r +	Relapse
Cured	7	5	8–69	3/12–30	6 × 50 2 10 × 50	4.4.4.5.6 7 (3 × 125) 3 × 250*	—	4 × 75[3] 3 3 × 150 3 × 150	1 *2 × 200
Improved	7	7	4–73	7/12–30	3 4.4.6 × 50	4.6.6.8 × 75 5 4 × 100	6.7.8 × 50 3	4.4 × 75 3 3 × 300	All 5
Unchanged	2	1	24–64	2–7	8 × 25 (2) 2 6 × 50	5 × 100 1	—	—	—
Total	16	13	4–73	3/12–30	7	13	3	6	6

TABLE XII.—DERMATITIS INFECTIOSA (38 CASES)

Result	Sex M	Sex F	Age incidence	Duration	85 kV. 25 r to 50 r	85 kV. 60 r to 75 r	140 kV. 25 r to 50 r	140 kV. 75 r	Subacute	Chronic	Relapse
Cured	12	3	18–74	3/12–7	4 6 6 5 8 8	5 6 2	8 8 5 6 8	8 3 8 8*	7	8	5 1*
Improved	20	3	21–71	2/12–15	3.4 4 6 6 10 7.4 12.4 11	5 2 5	6 4.4 8 6.8 8 9.11	4 3 4 6	8	15	——c 6
Unchanged	—	—	—	—	—	—	—	—	—	—	—
Total	32	6	18–74	2/12–15	15	4	13	6	15	23	7

TABLE XIII.—DERMATITIS VENENATA (23 CASES)

Result	Sex M	Sex F	Age incidence	Duration	85 kV. 50	85 kV. 75 to 100	140 kV. 50	140 kV. 75 +	Sub-acute	Chronic	Relapse
Cured	3	6	18–52	4/12–10	11 1	4 4 4 5 4	4 1*	6 × 75 4 × 150 3 3 × 200	3	6	1*
Improved	5	6	17–73	3/12–10	2* 4* 3 4 6	2 4*	5 3 6 7*	4 × 75 8 × 150* 3 5 × 100	5	6	5* F
Unchanged	—	3	24–50	5/12–10	6 2 6	—	—	3 × 75 1	1	2	—
Total	8	15	17–73	3/12–10	6	6	4	7	9	14	6

TABLE XIV.—SENSITIZATION DERMATITIS (62 CASES)

Result	Sex M	Sex F	Age incidence	Duration	85 kV. 12·5	25 | 50	75 100	125 150	140 kV. 25	50	75 100	150 200	Subacute	Chronic	Relapse
Cured	12	15	16–73	2/12–10	12	6 9	4(5) 6 9 7	4 4	8 11	4 6 6	4 4 6 .... 4 3 4 4 4	3 9	5	22	4 × 75 (2) 4 × 75 (2)
Improved	20	8	17–77	2/12–14	8	4 5 6 6 9 7	4 6 6 8 .... 5 11	3 4 4 8	6	4.4 6.8 8.10 8	4 6	3 .... 3 4	8	20	6
Unchanged	2	5	17–70	6/12– 4	1	6	4 3	.... 4 3	3	6 11 12	—		1	6	—
Total	34	28	17–77	2/12–14	11		21		16		13		14	48	10

[One "shower bath"]

[One "shower bath"]

Lastly Table XIV deals with that local and general reaction known as continuing sensitization dermatitis—one of the most troublesome problems with which dermatologists have to deal. In this group 62 cases have been treated. Once more there is greater response with the larger fractions in both 85 and 140 kV. but the best results obtained have been with the latter or larger kV., in which field no failures are noted and the ratio of cured to improved is 9 : 4.

Finally I would like to thank Dr. T. M. Young and the other members of Dr. Charteris's unit for their patient and painstaking help. My thanks are due also to Dr. J. F. Ferguson Smith, my senior registrar, and to Mr. James McCorquodale, my photographic technician.

KEY TO TABLES

Table V.—Small figures in treatment columns indicate number of treatments at stated dosage, e.g. 4×75^2 indicates 4 treatments of 75 r given twice. U = unknown.

Table VI.—Single small figures indicate number of treatments at r dosage noted at top of column: where multiplication sign is used as in figure 11, e.g. 6 × 12 indicates 6 cases had 12 treatments of 100. Details of 37 improved cases treated with 100 r are indicated at the foot of table.

Table VII.—Small figures in brackets indicate number of treatments and the fractions the time interval over which treatment was spread, e.g. good response, figure 1, 850 (5) 18/12 indicates a total of 850 r given in 5 treatments over eighteen months.

Table VIII.—As in Table VI but fractions in relapsed improved column indicate 7 cases out of column marked 11 and 2 cases out of column marked 6.

Table XI.—As Table VI.

Table XII.—Single figures as in Table VI but figures are divided into left and right columns corresponding to the smaller and the greater dosage indicated at top of columns, e.g. cured column, figure 5, small 6 right-hand side indicates 6 × 50 r. c = chronic.

Table XIII.—As for Table XII—improved relapsed column small F = female.

Table XIV.—As in Table XII with in addition dotted line separating upper and lower dosage levels as indicated in treatment columns.